

ბინირებული გამოყენებით და Medlung Ltd ენდობრონ-ქული სარქველებით. ცხოვრების ხარისხი შეფასდა სენტ ჯორჯის რესპირაციული (Saint George Respiratory Questionnaire (SGRQ) კითხვარით.

ბრონქოპლევრული მარგული საკვლევი ჯგუფში დაიხურა 30-დან 28 შემთხვევაში, ხოლო საკონტროლო ჯგუფში - 28-დან 19 შემთხვევაში. შესაბამისად, რეციდივების რაოდენობამ საკვლევი ჯგუფში შეადგინა 2 (6.7%), ხოლო საკონტროლო ჯგუფში - 9 (32.1%). ჯგუფებს შორის განსხვავება სტატისტიკურად სარწმუნოა ($\chi^2=6.1163$; $p=0.0134$). წინასაოპერაციო პერიოდის ხანგრძლივობამ საკონტროლო ჯგუფში შეადგინა 11.4 (SD17.0) დღე. წინა-სპ პერიოდის ხანგრძლივობა სარწმუნოდ მცირე იყო საკვლევი ჯგუფში - 0.9 (SD1.5) დღე ($p<0.001$); სტაციონარში დაყოვნების საშუალო მანძილებელმა საკვლევი ჯგუფში შეადგინა 8.0 (SD 14.1) დღე, შესაბამისმა მანძილებელმა საკონტროლო ჯგუფში შეადგინა 36.9 (SD 47.4) დღე (განსხვავება სარწმუნოა $p=0.0023$). პოსტინტერვენციული პლევრის დრუს დრენირების საშუალო ხანგრძლივო-

ბა საკვლევი ჯგუფში სპ-ის თერაპიის შემდგომ იყო - 2.6 (SD 1.7) დღე. საკონტროლო ჯგუფში კი 18.4 (SD 20.2) დღე ($p=0.0001$). ცხოვრების ხარისხის კვლევამ SGRQ კითხვარით აჩვენა, რომ ჯამური SGRQ ქულა საკვლევი ჯგუფში ინტერვენციის ჩატარებიდან 6 თვის შემდეგ სარწმუნოდ შემცირდა 65.4-დან 42.3-მდე ($p<0.001$), ე.ი. 23.1-ით. ჯამური SGRQ ქულა საკონტროლო ჯგუფში არასარწმუნოდ შემცირდა 63.6-დან 58.4-მდე ($p=NS$). სიმპტომების დომენის დინამიკა იყო სარწმუნოდ -27.6 ($p=0.008$), აქტივობების დომენის -31.5 ($p<0.001$) და გაეყვანების დომენის -17.0 ($p=0.011$).

სპ თერაპიამ გამოავლინა მაღალი ეფექტურობა ბრონქოპლევრული მარგულის კომპლექსურ მკურნალობაში ტრადიციულ მეთოდებთან (პლევრის დრუს პუნქცია და დრენირება) და ქირურგიულ მკურნალობასთან შედარებით, რაც გამოიხატა რეციდივების სიხშირის, პლევრული დრუს დრენირების ხანგრძლივობისა და პოსტიპტალში დაყოვნების სარწმუნოდ შემცირებაში.

DIRECT-ACTING ANTIVIRALS FOR HEPATITIS C DO NOT AFFECT THE RISK OF DEVELOPMENT OR THE OUTCOME OF HEPATOCELLULAR CARCINOMA

¹Gogichaishvili L., ¹Lobjanidze G., ²Tsertsvadze T., ³Chkhartishvili N., ³Jangavadze M.

¹Ivane Javakhishvili Tbilisi State University, Faculty of Medicine; ²Infectious Diseases, AIDS and Clinical Immunology Research Center, Tbilisi; ³Aleksandre Natishvili Institute of Morphology, TSU, Tbilisi, Georgia

HCV infection and its complications (hepatocellular carcinoma, decompensate cirrhosis) is a substantial public health burden. According WHO's Global Hepatitis Report [17], there are 1.7 million newly register cases every year and 400 000 death by related complications. Among them 137000 is a liver cancer patients, developed secondary after HCV infection [25]. Hepatocellular carcinoma (HCC) is an aggressive disease. It is the sixth most commonly diagnosed tumor and the fourth leading cause of cancer death in the world (841,000 new cases, 782,000 deaths annually). Incidence of liver cancer in male is 3 times higher. Therefore, it ranks second in terms of cancer deaths for males [1].

One of the main causes of the hepatocellular carcinoma (HCC) is a chronic hepatitis C induced liver cirrhosis. Effects of the different anti-hepatitis C virus treatment options on the epidemiology of HCC and its prognosis is conflicting and not fully understood [23]. Several author reports reduced risk of HCC during viral eradication therapy by Interferon (IFN)-based regimens, but some researchers show increasing incidence of the HCC recurrence after direct-acting antiviral (DAA) treatment, however these controversies based on different treatment options, measured parameters and observed groups and mainly derived from small series and observational studies [9,12,15,16,21,22].

In 2015 "Nationwide hepatitis C elimination program" was launched in Georgia. According the protocol, patients with HCC also receives antiviral treatment [8,13]. Based on this 5-year experience, we study effect of the different DAA therapy regimens on the incidence or recurrence of HCC and its prognosis.

Material and methods. Research [# Phd_F_17_33] has been

supported by Shota Rustaveli National Science Foundation of Georgia (SRNSFG). The protocol had obtained approval from the Ethics Committee of TSU Aleksandre Natishvili Institute of Morphology and complied with the ethical guidelines of the 2013 Declaration of Helsinki (WMA - declaration of Helsinki – ethical principles for medical research involving human subjects). All patients gave their written informed consent to participate in the study.

Overall, 408 patients were recruited in Georgian-French Joint Hepatology Clinic HEPA between April 2015-March 2016. The selection criteria were as follows: 1. age 50-65 years; 2. liver fibrosis level F3-F4 or cirrhosis at least 15 years of disease history; 3. HCV positive diagnosed by PCR method, whatever the level of viral load and genotype; 4. absence of previous complications of cirrhosis (ascites, gastrointestinal bleeding or HCC); 5. Child-Pugh class A or B; and 6. absence of severe extrahepatic disease. Their past medical history, essential clinical and biological parameters were recorded.

Clinical monitoring and management of adverse events were performed at regular base. The patients were seen by physicians every 6 months, and the essential clinical and biological data were recorded. Ultrasound examinations with Doppler were performed every 6 months. If mass lesions were detected by ultrasound, a diagnostic procedure using contrast-enhanced imaging (computed tomography scan or magnetic resonance imaging) and/or guided biopsy was performed according to the 2011 American Association for the Study of Liver Diseases

Guidelines [2]. A diagnosis of HCC was thus established by either histological examination or based on noninvasive crite-

ria (dynamic imaging revealing early arterial hypervascularization and portal washout) according to the different time periods. Treatment of HCC was determined using a multidisciplinary approach according to the European Association for the Study of the Liver–European Organisation for the Research and Treatment of Cancer and American Association for the Study of Liver Diseases guidelines for HCC [6].

All patients included in the study received anti-HCV treatment with direct-acting antivirals (DAAs) within the national hepatitis C elimination program in accordance with national protocols [24]. During April 2015–March 2016 treatment was provided with sofosbuvir (SOF) in combination with ribavirin (RBV), with or without pegylated interferon (IFN). Since March 2016, ledipasvir/sofosbuvir (LDV/SOF) was prescribed to all patients with or without RBV depending on the HCV genotype, level of fibrosis and previous treatment experience.

Outcomes of interest included 1) incidence of HCC and 2) mortality from HCC. To assess the incidence of HCC patients were followed since the date of enrollment in national hepatitis C elimination program until December 31, 2019 or date of HCC diagnosis whichever occurred first. Analysis of HCC incidence included patients who developed HCC after completing HCV treatment. In sensitivity analysis we further excluded patients who developed HCC within 12 months of starting anti-HCV treatment, because of high probability that these patients had already had undiagnosed HCC at the time of treatment initiation. To assess the mortality rate from HCC patients were followed since the date of HCC diagnosis until December 31, 2019 or death whichever occurred first. Incidence and mortality rates per 100 person-years (PY) of follow-up were calculated as number of events divided by

the total person-years of observation multiplied by 100. Bivariate comparisons were tested by Pearson’s chi-square or Fisher exact test as appropriate. Factors associated with the occurrence of HCC were evaluated in Cox proportional hazards regression model. All analyses were conducted using SAS v9.4. P values of <0.05 were considered statistically significant.

Results and discussion. Study included 408 HCV patients with advanced liver fibrosis defined as $\geq F3$ score by Metavir. The median age was 55 (interquartile range [IQR]: 52–59) years and 77.9% were men. All patients cured HCV infection with DAA treatment, 53.9% were treated with LDV/SOF \pm RBV (Table 1).

Overall, 54 (13.2%) patients had ever been diagnosed with HCC, including 7 persons who had been diagnosed before start of the HCV treatment. People with HCC were older (median 57 vs. 55 years old, $p=0.01$). No statistically significant differences were observed in terms of gender, HCV treatment regimen and duration (Table 1).

The incidence of HCC was quantified among 401 patients, who did not have HCC at the time of HCV treatment initiation. Patients were followed for the median 3.8 (IQR: 2.9–4.2) years contributing to 1393 PY of observation. A total 47 (11.2%) persons developed HCC at incidence rate of 3.37 per 100 PY (95% CI: 2.48–4.49). No statistically significant differences were found in the incidence rates by gender, HCV treatment regimen and duration (Fig. 1). In sensitivity analysis, which excluded persons who were diagnosed with HCC ($n=396$), the overall incidence was 3.02 per 100 PY (95% CI: 2.18–4.08), with no statistically significant differences found between the various groups (Fig. 2).

Table 1. Characteristics of study population ($n=408$)

	All ($n=408$)	No HCC ($n=354$)	HCC ($n=54$)	p value
Age, median years (IQR)	55 (52–59)	55 (52–58)	57 (53–62)	0.01
Sex, n (%)				
Men	318 (77.9)	76 (84.4)	14 (15.6)	0.46
Women	90 (22.1)	278 (87.4)	40 (12.6)	
HCV treatment regimen, n (%)				
LDV/SOF \pm RBV	220 (53.9)	190 (86.4)	30 (13.6)	0.94
SOF/RBV	114 (27.9)	100 (87.7)	14 (12.3)	
SOF/RBV + IFN	74 (18.1)	64 (86.5)	10 (13.5)	
HCV treatment duration, n (%)				
12 weeks	215 (52.7)	188 (87.4)	27 (12.6)	0.81
20 weeks	24 (5.9)	21 (87.5)	3 (12.5)	
24 weeks	153 (37.5)	130 (85.0)	23 (15.0)	
48 weeks	16 (3.9)	15 (93.7)	1 (6.3)	

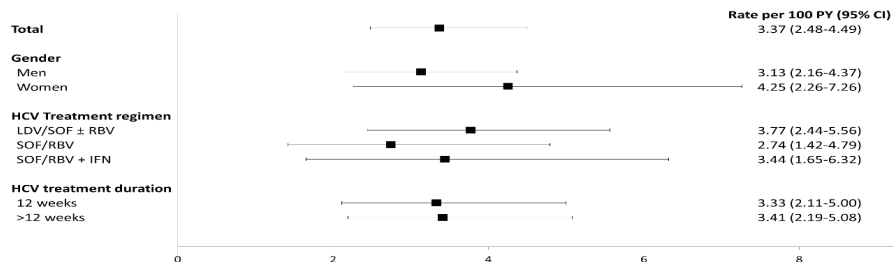


Fig. 1. Incidence of HCC among patients diagnosed any time after starting DAAs ($n=401$)

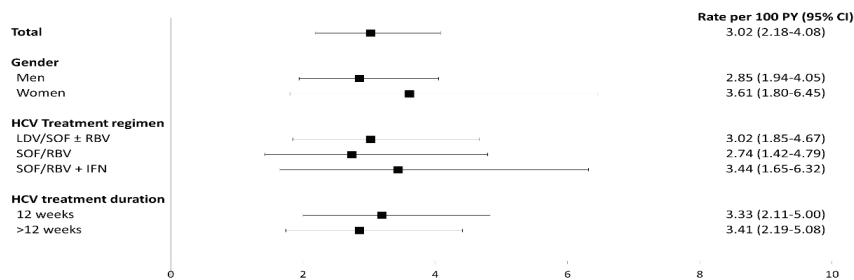


Fig. 2. Incidence of HCC among patients diagnosed at least 12 months after starting DAAs (n=396)

Table 2. Factors associated with HCC diagnosis in multivariate analysis

	Multivariate analysis among patients diagnosed with HCC any time after starting DAAs (n=401)				Multivariate analysis among patients diagnosed with HCC ≥12 months after starting DAAs (n=401)			
	All (n=401)	HCC (n=47)	HR (95% CI)	p value	All (n=396)	HCC (n=42)	HR (95% CI)	p value
Age, median years (IQR)	55 (52-59)	56 (53-61)	1.08 (1.01-1.15)	0.03	55 (52-59)	55 (52-58)	1.08 (1.01-1.16)	0.03
Sex, n (%)								
Women	89 (22.2)	13 (14.6)	1		87 (22.0)	11 (12.6)	1	
Men	312 (77.8)	34 (10.9)	0.87 (0.45-1.68)	0.67	309 (78.0)	31 (10.0)	0.94 (0.46-1.93)	0.87
HCV treatment regimen, n (%)								
LDV/SOF ± RBV	215 (53.6)	25 (11.6)	1		210 (53.0)	20 (9.5)	1	
SOF/RBV	112 (27.9)	12 (10.7)	0.58 (0.27-1.26)	0.17	112 (28.3)	12 (10.7)	0.81 (0.35-1.84)	0.61
SOF/RBV + IFN	74 (18.5)	10 (13.5)	1.08 (0.49-2.49)	0.86	74 (18.7)	10 (13.5)	1.14 (0.48-2.68)	0.77
HCV treatment duration, n (%)								
12 weeks	211 (52.6)	23 (10.9)	1		210 (53.0)	22 (10.5)	1	
>12 weeks	190 (47.4)	24 (12.6)	1.31 (0.62-2.80)	0.48	186 (47.0)	20 (10.8)	0.98 (0.43-2.24)	0.96

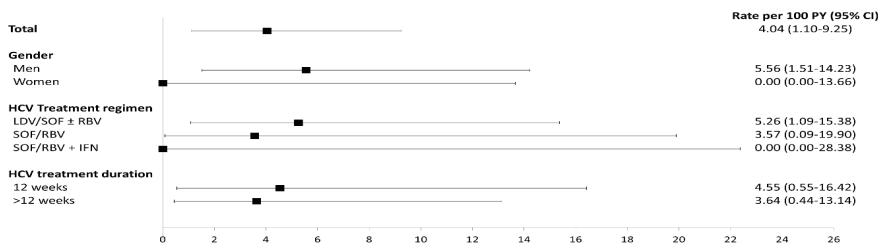


Fig. 3. Mortality rates among patients with HCC (n=54)

In multivariate model, age per year increase was the only factor associated with the diagnosis of HCC: hazard ratio (HR) 1.08 (95% CI: 1.01-1.15, p=0.03). Similar results were obtained in sensitivity analysis (Table 2).

Mortality was assessed among 54 persons with diagnosed HCC. They were followed for the median 1.6 (IQR: 1.0-4.2) years contributing to 99 PY of observation. A total of 4 (7.4%) with mortality rate of 4.04 per 100 PY (95% CI: 1.10-9.25). All deceased patients were men, however no statistically significant differences were observed neither by gender nor by HCV treatment regimen and duration (Fig. 3).

Mortality was assessed among 54 persons with diagnosed HCC. They were followed for the median 1.6 (IQR: 1.0-4.2) years contributing to 99 PY of observation. A total of 4 (7.4%) with mortality rate of 4.04 per 100 PY (95% CI: 1.10-9.25). All deceased patients were men, however no statistically significant differences were observed neither by gender not by HCV treatment regimen and duration (Fig. 3).

After successful implementation of “Nationwide hepatitis C elimination program” in Georgia, main treatment modality

for HCV patients became DAA therapy. Sustained viral response (SVR) rate after DAA antiviral treatment is significant and reaches 98% of patients with compensated liver disease, however, patients with decompensated cirrhosis and patients with HCV genotype 3 infection SVR rates are 85% to 95% [4]. It is well established that, SVR is associated with a 71% reduction of HCC risk [10]. Despite this advantages, there are number of papers which indicates increased risk of HCC after DAA treatment with comparison of other treatment modalities [3, 18, 20, 26]. All such research had their limitations therefore it is highly important to accumulate more evidence in this field. Possible mechanisms of increasing of HCC risk after use of DAA was suggested not only direct cancerogenic effect of this drugs, but also impairment of immune tumor surveillance [14, 19]. However, this hypothesis is rejected by recent studies, which showed pre-treatment modification of the Cytokines and suggesting that immune-modulation by DAA does not trigger HCC development [5]. These findings correlates with serum miRNA levels in HCC patients (our unpublished data).

Our study was limited by relatively small number of patients and short follow-up time. Also, some treatment groups were limited because of their rarity in practice. We use targeted patient groups, with high risk of HCC, to revile changes in study parameters after DAA treatment. We selected patients with higher risk of HCC (age 50-65 and liver fibrosis level F3-F4), which gave us more chance to identify difference between groups.

We found no statistically significant differences between treatment groups. They equally affect HCC development risk and mortality due cancer in short- and medium-term prognosis. Also, treatment duration did not change HCC risk. This findings correlates with other studies which includes larger but more varied populations [14]. However, based on our data, we cannot exclude the possibility that DAA can change HCC risk and cancer mortality in long-term prognosis [7]. We need more follow-up time to identify this probability.

Our study couldn't directly answer question, whether DAA treatment changes the HCC risk relatively with NO- or interferon-based regimen treated patients. Study design with "no treatment" patients would be unethical. Also, DAA treatment shows superiority with interferon-based regimens, and thus IFN cannot be given as a single medication for HCV treatment. Moreover, large retrospective cohort studies show no difference in HCC incidence rates between DAA and IFN-only treatment regimens [10,11].

In conclusion, we find that neither different DAA regimens nor different treatment duration affects HCC risk after antiviral treatment. Moreover, there is not significant changes in mortality rate due to HCC in this groups. It can be concluded therefore, that HCC status is not contraindication for DAA treatment, spatially at early stages of cancer, when tumor is curative. Our results show, that also long duration DAA treatment can be safely used.

Acknowledgements. This research has been supported by Shota Rustaveli National Science Foundation of Georgia (SRN-SFG) - (Grant N [# PhD_F_17_33] - Outcome, risk of development and new highly sensitive blood circulating tumour markers of a Hepatocellular Carcinoma in the course of Ledipasvir/Sofosbuvir treatment of HCV infected patients.) Authors are grateful to Georgian-French Joint Hepatology Clinic HEPA team for great support in clinical investigation.

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SUMMARY

DIRECT-ACTING ANTIVIRALS FOR HEPATITIS C DO NOT AFFECT THE RISK OF DEVELOPMENT OR THE OUTCOME OF HEPATOCELLULAR CARCINOMA

¹Gogichaishvili L., ¹Lobjanidze G., ²Tsertsvadze T., ²Chkhartishvili N., ³Jangavadze M.

¹Ivane Javakhishvili Tbilisi State University, Faculty of Medicine; ²Infectious Diseases, AIDS and Clinical Immunology Research Center, Tbilisi; ³Aleksandre Natishvili Institute of Morphology, TSU, Tbilisi, Georgia

HCV infection and its complications, especially hepatocellular carcinoma, is a substantial public health burden. In 2015 “Nationwide hepatitis C elimination program” was launched in Georgia. According to the protocol, patients with HCC also receive DAA antiviral treatment. We study the effect of the different DAA therapy regimens on the incidence or recurrence of HCC and its prognosis.

Overall, 408 patients were recruited in Georgian-French Joint Hepatology Clinic HEPA between April 2015-March 2016. The selection criteria were as follows: 1 - age 50-65 years; 2. Liver fibrosis level F3-F4 or cirrhosis at least 15 years of disease history; 3. HCV positive diagnosed by PCR method, whatever the level of viral load and genotype; 4. absence of previous complications of cirrhosis (ascites, gastrointestinal bleeding or HCC); 5. Child-Pugh class A or B; and 6. absence of severe extrahepatic disease. Essential clinical and biological parameters were recorded. Clinical monitoring and management of adverse events were performed on a regular base. HCV All patients included in the study received anti-HCV treatment with direct-acting antivirals (DAAs) within the national hepatitis C elimination program in accordance with national protocols. During April 2015-March 2016 treatment was provided with sofosbuvir (SOF) in combination with ribavirin (RBV), with or without pegylated interferon (IFN). Since March 2016, ledipasvir/sofosbuvir (LDV/SOF) was prescribed to all patients with or without RBV depending on the HCV genotype, level of fibrosis, and previous treatment experience.

In conclusion, we find that neither different DAA regimens

nor different treatment duration affects HCC risk after antiviral treatment. Moreover, there are no significant changes in mortality rate due to HCC in these groups. Therefore, it can be concluded, that HCC status is not a contraindication for DAA treatment, especially at the early stages of cancer, when a tumor is curative.

Keywords: Direct-acting antiviral, hepatitis C, hepatocellular carcinoma.

РЕЗЮМЕ

ВЛИЯНИЕ ПРОТИВОВИРУСНЫХ ПРЕПАРАТОВ ПРЯМОГО ДЕЙСТВИЯ НА РИСК РАЗВИТИЯ И ИСХОД ГЕПАТОЦЕЛЛЮЛЯРНОЙ КАРЦИНОМЫ ПРИ ГЕПАТИТЕ С

¹Гогичаишвили Л.Т., ¹Лобжанидзе Г.В., ²Цертсвадзе Т.Н., ²Чхартишвили Н.И., ³Джангавадзе М.Б.

¹Тбилисский государственный университет им. И. Джавахишвили, медицинский факультет; ²Научно-исследовательский центр по инфекционным болезням, СПИДа и клинической иммунологии; ³Институт морфологии им. А. Нативили, Тбилисский государственный университет, им. И. Джавахишвили Грузия

Вирусная инфекция гепатита С (ВГС) и ее осложнения, особенно гепатоцеллюлярная карцинома (ГЦК), являются актуальной проблемой общественного здравоохранения. В 2015 г. в Грузии запущена «Общенациональная программа элиминации ВГС». Согласно протоколу, пациенты с ГЦК получают противовирусное лечение прямого антивирусного действия (ПАД). Нами изучено влияние различных режимов ПАД-терапии на частоту и/или рецидив ГЦК и его прогноз.

В период с апреля 2015 г. по март 2016 г. в Грузино-французской совместной гепатологической клинике HEPA наблюдались 408 пациентов. Критериями включения в исследование явились: возраст 50-65 лет; уровень фиброза печени F3-F4 или цирроз печени в течение не менее 15 лет; ВГС-положительный диагноз методом полимеразной цепной реакции; отсутствие осложнений цирроза печени (асцит, желудочно-кишечные кровотечения или ГЦК); класс А или В по шкале Чайлд-Пью; отсутствие тяжелых внепеченочных заболеваний. Зарегистрированы основные клинические и биологические параметры. Все пациенты, включенные в исследование, получали противовирусное лечение ВГС ПАД-ом в рамках Национальной программы элиминации вируса гепатита С в соответствии с национальным протоколом - софосбувир (SOF) в комбинации с рибавирином (RBV), с или без пегилированного интерферона (IFN). С марта 2016 г. ледипасвир/софосбувир (LDV/SOF) назначается всем пациентам с или без RBV с учетом генотипа ВГС, уровня фиброза и предыдущей тактики лечения.

Проведенные исследования показали, что ни схема, ни продолжительность лечения ПАД не влияют на риск развития ГЦК после противовирусного лечения. Более того, в исследуемых группах статистически значимых изменений уровня смертности от ГЦК не выявлено. Таким образом, следует заключить, что статус ГЦК не является противопоказанием для лечения ПАД, особенно на ранних стадиях рака, когда опухоль излечима.

რეზიუმე

ჰეპატიტ C ვირუსის საწინააღმდეგო პირდაპირი მოქმედების ანტივირუსული პრეპარატების გავლენის განსაზღვრა ჰეპატოცელულური კარცინომის განვითარების რისკსა და გამოსავალზე

¹ლ.გოგიანიშვილი, ¹გ.ლობუანიძე, ²თ.ცერცვაძე, ²ნ.ხარტიშვილი, ³მ.ჯანგაგაძე

¹ი. ჯავახიშვილის სახ. თბილისის სახელმწიფო უნივერსიტეტი, მედიცინის ფაკულტეტი; ²ინფექციური პათოლოგიის, შიდსისა და კლინიკური იმუნოლოგიის სამეცნიერო-პრაქტიკული ცენტრი; ³ა. ნათიშვილის სახ. მორფოლოგიის ინსტიტუტი, თბილისის სახელმწიფო უნივერსიტეტი, საქართველო

C-ჰეპატიტის ვირუსით ინფექცია (HCV) და მისი გართულებები, განსაკუთრებით ჰეპატოცელულური კარცინომა (HCC), წარმოადგენენ საზოგადოებრივი ჯანდაცვის მნიშვნელოვან ტვირთს. საქართველოში 2015 წლიდან დაიწყო HCV ელიმინაციის ეროვნული პროგრამა. პროტოკოლის თანახმად, დღეისათვის HCC-ით დაავადებულ პაციენტებსაც უტარდებათ პირდაპირი მოქმედების ანტივირუსული პრეპარატებით (DAA) მკურნალობა. ჩვენს მიერ შესწავლილია სხვადასხვა DAA მკურნალობის რეჟიმების ზემოქმედება HCC-ს ინციდენტობაზე, რეციდივსა და პროგნოზზე.

კვლევისათვის შერჩეული იყო 408 პაციენტი (ქართულ-ფრანგული ერთობლივი ჰეპატოლოგიური კლინიკა „ჰეპა“) 2015 წლის აპრილიდან 2016 წლის

მარტის ჩათვლით. შერჩევის კრიტერიუმები იყო: ასაკი 50-65 წელი; დეიდლის ფიბროზის დონე F3-F4 ან ციროზი მინიმუმ 15 წლის ხანდაზმულობით; HCV დადებითი PCR მეთოდით, ვირუსის ნებისმიერი რაოდენობით და/ან გენოტიპით; წინა პერიოდში ციროზის გართულებების არარსებობა (ასციტი, გასტროინტესტინური სისხლდენა და/ან HCC); Child-Pugh შკალით A და/ან B კატეგორია; მძიმე ექსტრაჰეპატური დაავადებების არარსებობა. პაციენტებს რეგულარულად უტარდებოდათ მონიტორინგი და კლინიკური მართვა. კვლევაში ჩართულ ყველა პაციენტს ანტი HCV მკურნალობა უტარდებოდა პირდაპირი მოქმედების ანტივირუსული პრეპარატებით (DAA) C ვირუსული ჰეპატიტის ელიმინაციის ეროვნული პროგრამის ფარგლებში ნაციონალური პროტოკოლის მიხედვით: 2015 წლის აპრილიდან 2016 წლის მარტის ჩათვლით პაციენტებს მკურნალობა უტარდებოდათ სოფოსბუვირით (SOF) კომბინაციაში რიბავირინთან (RBV), პეგილირებულ ინტერფერონთან (IFN) ან მის გარეშე. 2016 წლის მარტიდან ყველა პაციენტს დაენიშნა ლედიპასვირი/სოფოსბუვირი (LDV/SOF) RBV-სთან ერთად ან მის გარეშე. გენოტიპის, ფიბროზის ხარისხისა და მანამდე არსებული მკურნალობის გამოცდილების გათვალისწინებით.

კვლევამ აჩვენა, რომ DAA-ს გამოყენებული რეჟიმები და ხანგრძლივობა არ იწვევს HCC-ი

ს რისკის მატებას. მეტიც, აღნიშნულ ჯგუფში HCC-ს გამო სიკვდილობის მატების სარწმუნო ცვლილება არ შეინიშნება. შესაბამისად, HCC-ას არსებობა DAA-ით მკურნალობისას არ წარმოადგენს წინააღმდეგგვენებას, განსაკუთრებით სიმსივნის ადრეულ სტადიაზე, სანამ სიმსივნე ითვლება განკურნებადად.

ВЛИЯНИЕ УРОВНЯ ПОТРЕБЛЕНИЯ АЛКОГОЛЯ НА ТЕЧЕНИЕ И ЭФФЕКТИВНОСТЬ ЛЕЧЕНИЯ ВПЕРВЫЕ ДИАГНОСТИРОВАННОГО ТУБЕРКУЛЁЗА ЛЁГКИХ

^{1,3}Грек И.И., ^{1,3}Рогожин А.В., ¹Кушнир В.Б., ²Колесникова Е.Н., ¹Кочуева М.Н.

¹Харьковская медицинская академия последипломного образования; ²ГУ «Национальный институт терапии им. Л.Т. Малой НАМН Украины»; ³Харьковский национальный университет им. В.Н. Каразина, Украина

По оценкам Всемирной организации здравоохранения (ВОЗ) в 2019 г. в мире зарегистрировано более 10,4 млн. случаев заболевания туберкулезом (ТБ) и около 1,6 млн. смертей вследствие этой инфекционной патологии [22]. Известно, что потребление алкоголя является одним из основных факторов риска развития ТБ и ухудшения прогноза терапии [5,9,17,22]. От 10% до 20% всех случаев смерти от ТБ во всем мире ассоциированы с употреблением алкоголя [9,23]. В 2017 г. одной из целевых задач ВОЗ являлась профилактика и лечение расстройств, связанных с потреблением алкоголя, как ключ к снижению глобальной заболеваемости и смертности от ТБ [22]. Распространенность расстройств, связанных с потреблением алкоголя, или злоупотреблением алкоголем среди пациентов с ТБ во всем мире колеблется от 15% до 70%. [10,13,16,19,20]. Лица, потребляющие алкоголь, имеют более неблагоприятные результаты лечения из-за влияния поведенческих (низкая мотивация и случаи уклонения от лечения [3,8,11]) и биологических механизмов (негативное влияние алкоголя на врожденные и адаптивные

иммунные реакции, защитную барьерную функцию легких, гепатобилиарную систему, абсорбцию и метаболизм лекарственных препаратов, направленных на терапию ТБ и вируса иммунодефицита человека (ВИЧ) [7,12,14,15].

Несмотря на распространенные данные об отрицательном влиянии злоупотребления алкоголем на течение туберкулезного процесса, по сей день не имеется информации о клеточно-молекулярных механизмах действия алкоголя на большинство ТБ и данных об изменениях клинико-лабораторного, иммунного и окислительного статусов в зависимости от уровня употребления алкоголя.

Целью исследования явилась оценка эффективности лечения после интенсивной фазы терапии у больных с впервые диагностированным туберкулезом лёгких в зависимости от уровня потребления алкоголя и разработка метода прогнозирования эффективности лечения.

Материал и методы. Обследованы 109 мужчин с инфильтративной формой впервые диагностированным туберкулезом лёгких (ВДТБЛ) в возрасте от 20 до 50 лет. Медиа-