INTRODUCTION
Chronic pancreatitis is very actual problem in modern medicine. In Ukraine the prevalence of GI disorders has been significantly increased during past few years. The prevalence of chronic pancreatitis from 2008 to 2012 has been increased by 2,2 times, the pancreas disorders - by 3,2 times. According to statistical data from Ministry of healthcare in Ukraine during the period from 2006 to 2013 the numbers of admissions with chronic pancreatitis was increased by 30,2%. The combination of few diseases has palace in the majority of cases in gastroenterology clinics. Chronic pancreatitis is one of the most common disorder that have been diagnosed with chronic hepatitis and liver cirrhosis.

In case of combination of liver and pancreas damage the prognosis of recovery is unfavorable due to the complicated treatment plan. Under the circumstances that have occurred during last decades: epidemic viral hepatitis, alcohol abuse, increased obesity and dysmetabolic syndrome prevalence as well as non-alcoholic fatty liver disease leading to increased amount of patients with terminal liver conditions and according to the epidemiological analysis it reached it’s peak in 2010-2020 [1].

During the past few years attention to the pathogen, that can cause the inflammation in pancreas, has increased. Hepatitis due to HCV is the progressive disease that leads to chronic active hepatitis, cirrhosis and hepatocellular carcinoma. Chronic hepatitis C may have persistent course with the minimum of clinical symptoms and biochemical changes causing late diagnostic of active process. Morphological changes in liver occur asymptomatically. Most often after the exposure to the pathogen it takes the long time to develop first signs of infection. Negative risk factors in natural course of disease include: age over 40 at the moment of exposure, non-Caucasian race, alcohol abuse, obesity, ferrous dysmetabolism an dysmetabolic syndrome [2].

Many people with Chronic hepatitis C develop diabetes mellitus type II. This prevalence is much higher compared with general population and with group of patients suffering from other chronic liver disorders, such as HBV infection, alcohol liver disease and primary biliary cirrhosis.

According to the experts around 160 billion people, 2,35 % of all world population are infected with HCV. Besides that, liver failure as a result of HCV infection is one of leading causes for liver transplantation. Usually immunological disorders are extra hepatic symptoms of chronic hepatitis C infection, however virus may have direct cytopathic effect and can infect many other tissue, besides liver [3].
the progression of liver disorder. Significant prevalence of HCV infection in population, high level of process chronization with cirrhosis formation and hepatocellular carcinoma development as well as variety of extra-hepatic symptoms determine topicality of the HCV problem. At the same time the insufficiency in evolution of primary and secondary preventions of chronic pancreatitis motivates to deeper discovery of it’s physiological mechanisms with concomitant chronic hepatitis C, as well as to new approaches to develop prevention, treatment and rehabilitation [4, 5].

THE AIM
To discover the clinical aspects of chronic pancreatitis course in patients with concomitant viral hepatitis C.

MATERIALS AND METHODS
57 patients with chronic pancreatitis and concomitant chronic hepatitis C (group I) were examined, they were under outpatient observation at the Ternopil Primary Care Center and admitted to gastrointestinal department at Ternopil Hospital №2. Comparison group consisted of 20 patients with chronic pancreatitis (group II). The ambulatory charts of the patient were the sources of information. To diagnose chronic pancreatitis and viral hepatitis C all patients were examined according to the protocols (Ministry of Healthcare in Ukraine orders №638 from 10.09.2014 and №729 from 18.07.2016). Average age of the patients was 53,5±22,5 years. Number of People in retirement prevailed. Diagnoses of chronic pancreatitis an viral hepatitis C were verified based on disease history, clinical symptoms as well as clinical and instrumental tests results. General clinical and biochemical tests were performed for patients with chronic pancreatitis in exacerbation phase and non-stable remission, for the patient with viral hepatitis C - in stable remission (ALT level remains normal). Results of coprological examination were analyzed in both groups. Each abnormal characteristic of coprogram was rated as one point with he following normal value: daily amount of stool - 120-200g, SAUSAGE-LIKE shape, homogeneous consistency, brown color, absence of undigested food, absence of mucus, blood and pus, normal flora, parasites-absent, ph-6-8, muscle fibers-absent, neutral fat-absent, fatty acids-few, starch and soup -small amount, undigested cellulose-few, oxalates-absent, stercobilin - small amount, bilirubin - absent. To interpret the result of Ultrasound diagnostic in points we use Marsel- Cambrige classification visualization criteria.

The arithmetic mean (M) with mean square deviation (m) were calculated. To verify statistical hypothesis we used parametric and nonparametric methods. In parametric distribution we used Student’s T-criteria, in nonparametric-Manna-Uitni’s U-criteria. For statistical hypothesis verification null hypothesis was rejected at significance value p<0,05.

RESULTS
Pain, dyspeptic syndromes and defecation disturbances take the major place in clinical course of chronic pancreatitis with concomitant viral hepatitis C. These symptoms were more severe compared with patients who suffered from isolated chronic pancreatitis (see table I).

In patient with chronic pancreatitis and concomitant viral hepatitis C mild anemia with decreased level of albumin, hyperbilirubinemia, hypercholesterolemia, elevated level of transaminases, amylase and urine diastase were detected (see table II).

Differences in value results reflect multiple dysmetabolic disorders in patient with chronic pancreatitis and concomitant viral hepatitis C, which were more reliably significant compared with group II and have shown the aggravating role of concomitant viral hepatitis C on laboratory characteristics in patients suffering from chronic pancreatitis.

In analysis of patient’s coprogram with chronic pancreatitis and concomitant viral hepatitis C in comparison with group of patients who suffer from isolated chronic pancreatitis we noticed more severe pathological changes that have proved negative effect of viral hepatitis C on clinical course of chronic pancreatitis. It was established based on worse value results , more expressed inflammation syndrome and signs of dysbacteriosis, which reflect more severe pancreas exocrine function disorder in case of combination of chronic pancreatitis and viral hepatitis C. The analysis of Ultrasound diagnostic results in both groups has shown: the depth of structural changes in patient with chronic pancreatitis and concomitant viral hepatitis C was 4,56±0.55 point, that reflect the moderate severity. In patients with isolated chronic pancreatitis - 1,900±0,3 points. (see fig. 2).

Compression of these two parameters (p<0,05) allowed us to show the contrast of more severe structural changes in pancreas (according to Ultrasound tests results data) in patients with concomitant viral hepatitis C.

DISCUSSION
Beyond the liver, HCV chronic infection leads to a multifaceted systemic disease. Some extrahepatic manifestations are immune mediated while others seem to be driven by chronic inflammation. Such extrahepatic manifestations should be well known by clinicians. They should have an impact on the care of patients with HCV infection. So far more than 30 different conditions have been associated with chronic HCV infection. In general, the appearance of extrahepatic manifestations of HCV infection is unpredictable, that is, independent of the
stage of the liver disease. A clear association with chronic hepatitis C has been established for many of these conditions, while, for some diseases, good-quality evidence linking them to HCV infection is still missing. [7] Finally,

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**Table I. Main Gastrointestinal syndromes in groups of patients with Chronic pancreatitis depending on presence of concomitant Viral hepatitis C (M+m)**

<table>
<thead>
<tr>
<th>Clinical syndrome and symptom</th>
<th>Group I (n=57)</th>
<th>Group II (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain syndrome</td>
<td>100,0 %*</td>
<td>80,0 %</td>
</tr>
<tr>
<td>Dyspeptic syndrome</td>
<td>87,5 %*</td>
<td>70,0 %</td>
</tr>
<tr>
<td>Defecation disorders</td>
<td>89,4 %*</td>
<td>65,0 %</td>
</tr>
<tr>
<td>Constipation</td>
<td>33,3 %*</td>
<td>25,0 %</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>26,8 %*</td>
<td>15,0 %</td>
</tr>
<tr>
<td>Alternation of constipation and diarrhea</td>
<td>45,6 %*</td>
<td>45,0 %</td>
</tr>
</tbody>
</table>

Note: Probability of value difference in group I in relation to group II (p<0,05).

**Table II. Main clinical and biochemical characteristics of blood in both groups (M+m)**

<table>
<thead>
<tr>
<th>Laboratory characteristic</th>
<th>Group I (n=57)</th>
<th>Group II (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythrocyte, women, /l</td>
<td>4,02±0,33*</td>
<td>4,48±0,11</td>
</tr>
<tr>
<td>Hemoglobin, women, g/l</td>
<td>117,0±10,04*</td>
<td>132,0±9,48</td>
</tr>
<tr>
<td>Erythrocyte, men, /l</td>
<td>4,4±0,2*</td>
<td>4,58±0,13</td>
</tr>
<tr>
<td>Hemoglobin, men, g/l</td>
<td>140,0±7,6*</td>
<td>154,0±9,43</td>
</tr>
<tr>
<td>Leucocytes /l</td>
<td>5,34±1,13*</td>
<td>5,27±1,06</td>
</tr>
<tr>
<td>ESR mm/hr</td>
<td>9,0±5,59*</td>
<td>8,0±5,47</td>
</tr>
<tr>
<td>Total bilirubin mmol/l</td>
<td>29,6±11,28*</td>
<td>15,83±3,76</td>
</tr>
<tr>
<td>Total protein g/l</td>
<td>58,02±6,67*</td>
<td>82,6±3,54</td>
</tr>
<tr>
<td>Total cholesterol mg/dL</td>
<td>5,52±0,83*</td>
<td>4,4±0,56</td>
</tr>
<tr>
<td>ALT IU/L</td>
<td>1,36±0,51*</td>
<td>0,47±0,14</td>
</tr>
<tr>
<td>AST IU/l</td>
<td>0,66±0,14*</td>
<td>0,26±0,11</td>
</tr>
<tr>
<td>Blood amylase mg/ hr*ml</td>
<td>38,97±9,08*</td>
<td>26,73±3,83</td>
</tr>
<tr>
<td>Urine diastase mg/ hr*ml</td>
<td>168,6±97,34*</td>
<td>98,96±58,12</td>
</tr>
</tbody>
</table>

Note: Probability of value difference in group I in relation to group II (p<0,05).

**Pic. 1. Total score of coprogram in points in both groups**
there are three manifestations classically considered as HCV-associated and whose relationship has not been established in the later publications: Thyroid manifestations (associated with interferon therapies and some age groups), pulmonary fibrosis and Mooren corneal ulcers. Finally, it is worth considering that the appearance of extrahepatic manifestations and positive immunological results can lead to the diagnosis of a specific system autoimmune disorder, according to the corresponding criteria. [8] Patients with CHC infection are at higher risk of developing CKD. Metabolic and cardiovascular factors need to be considered, as well as extrahepatic manifestations affecting the kidneys, in addition to the underlying liver disease. [9]

The extrahepatic manifestations of hepatitis C include effects on the central nervous system, which have been associated with the ability of hepatitis C virus (HCV) to replicate in microglial and endothelial cells and the chronic inflammation induced by HCV. HCV can induce impaired neurocognition, which is clinically manifested by impaired quality of life, fatigue, and brain fog. These cognitive defects can be present even in patients with mild histologic HCV and have been confirmed by neurocognitive testing and brain imaging by magnetic resonance spectroscopy. Neurocognitive defects include loss of functioning memory and subtle changes in attention and processing speed. [10]

CONCLUSIONS
Comparative analysis of clinical, laboratory and instrumental signs in patients with chronic pancreatitis depending on the presence of viral hepatitis C discovered the following aspects of this combination: more severe pain and dyspeptic syndromes, worsening of laboratory changes which include mild anemia, decreased blood albumin level, hyperbilirubinemia, hypercholesterolemia, elevated transaminases, blood amylase and urine diastase, increased total score of comprogram (more expressed inflammation syndromes and dysbacteriosis in colon), have shown higher level of pancreas exocrine function disorders as well as increased total score of Ultrasonic diagnostic tests chances to moderate level of severity according to Marsel-Cembridge classification.

REFERENCES

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According to the order of the Authorship.

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The Authors declare no conflict of interest.

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