INTRODUCTION
An estimated up to 3% of the world’s population is living with hepatitis C virus (HCV) infection [1]. Being one of the main causes of chronic liver disorders, such as progressive liver fibrosis, cirrhosis, liver failure, and hepatocellular carcinoma, HCV is also responsible for extrahepatic manifestations involving the skin, kidneys, salivary glands, eyes, thyroid, cerebrovascular, and immune systems [2-6]. The severity of liver damage due to the disease doesn’t correlate with extrahepatic manifestation [7] but the mortality rate is higher in HCV patients with extrahepatic complications [8-11]. However, little to no data are published about aseptic meningitis as an extrahepatic manifestation of hepatitis C in a young white adult.

THE AIM
We aimed to provide a clinical case presentation of aseptic meningitis as an extrahepatic manifestation of hepatitis C in a white young female European adult.

CLINICAL CASE
We have provided a complex clinical, neurological, laboratory, and instrumental analysis of an extrahepatic manifestation of hepatitis C in a white young female European adult.

VITALS UPON ADMISSION
Upon admission to the department, the patient showed a normal general condition. The respiratory rate was 16
breaths per minute, oxygen saturation was 99%, and blood pressure was 110/70 mm Hg. A heart rate of 70 beats per minute and a temperature of 36.6° C.

CLINICAL EXAMINATION
The skin and visible mucous membranes were clean, having a healthy appearance, pinkish. Moderate facial hyperemia. Peripheral lymph nodes were not palpable. Vesicular respiration in the lungs, no wheezing. The liver was slightly enlarged +2 cm. There were no traces of non-drug injections. No edema. The abdomen was soft and painless on palpation.

NEUROLOGICAL STATUS
The patient was conscious and available for productive contact. Understood the addressed language and followed instructions. Anxious, low mood. Own language was preserved. Meningeal sign represented questionable stiffness in the occipital muscles. Eye slits D = S, pupils D = S. Movements of the eyeballs were full. No diplopia. The face was symmetrical. Swallowing was not disturbed. The pharyngeal reflex and muscle tone in the extremities were preserved. Muscle strength in the extremities was diffusely reduced to 4.5 points. Tendon reflexes from the arms were high, and symmetrical, from the legs were high also, D = S. Left pathological carpal reflex was present. Surface sensitivity was not impaired. Pelvic functions with no issues. The patient was unsteady in Romberg’s posture but the gait was intact.

LABORATORY AND INSTRUMENTAL EXAMINATION RESULTS
MRI of the brain upon admission was without pathological changes. Cerebrospinal fluid on TORCH infection was negative. Cerebrospinal fluid analysis: protein 0.38, glucose - 2.80, cell count 120 (57% lymphocyte, 43% neutrophil), erythrocyte 8-10, light yellow color, slightly cloudy. Cerebrospinal fluid samples were found positive for HCV RNA by polymerase chain reaction. Bacteriological analysis of cerebrospinal fluid - negative. Express test on HCV - positive. Blood for borreliosis - negative.

Within three days after admission: General cerebrospinal fluid analysis: protein 0.69, glucose - 4.20, cell count 199 (98% lymphocyte, 2% neutrophil), erythrocyte 20-30, color light yellow, slightly cloudy; MRI of the brain: signs of intravenous hypotension.

DIAGNOSIS
Aseptic meningitis as an extrahepatic manifestation of hepatitis C with mild paresis of the third pair of the right cranio-cerebral nerve, cephalic, and asthenic syndrome.

DISCHARGE
After treatment, clinical and laboratory improvements were noted: no complaints were voiced, and meningeal signs were not detected. However, mononuclear pleocytosis of the cerebrospinal fluid and an increased ALT persisted. The patient was discharged from the hospital in good condition under the supervision of a neurologist and an infectious disease specialist at the place of residence.

DISCUSSION
HCV has a large public health impact across the world. The prevalence of infection is soaring in lower and middle-income countries. The highest is in Egypt 4.4-15.0%, Gabon 4.9-11.2%, Uzbekistan 11.3%, Cameroon 4.9-13.8%, Mongolia 9.6-10.8%, Pakistan 6.8%, Nigeria 3.1-8.4%, and Georgia 6.7% [16]. In contrast, in high-income countries, the prevalence of chronic HCV is below 2% [17, 18].

HCV infections are mainly caused by high-risk exposures and behaviors among specific populations (men who have sex with men, intravenous drug users, people with multiple sexual partners, people who had cosmetic practices such as tattooing, people who had blood transfusions, blood products, or organ donations, unsterile dental equipment, etc). No vaccine against hepatitis C exists and no effective pre- or post-exposure prophylaxis is available [1]. However, viral eradication reduces the rate of extrahepatic deaths significantly [19-21].

Reported extrahepatic manifestations of HCV represent a wide spectrum of disorders, such as mixed cryoglobulinemia, associated vasculitis with multi-organ-pathology, porphyria cutanea tarda, lichen planus, autoimmune and/or lymphoproliferative disorders, fatigue, depression, cognitive impairment, insulin resistance, diabetes mellitus, accelerated atherosclerosis, cardiovascular, renal, central nervous system diseases, and increased cardiovascular disease morbidity and mortality [22-26]. However, data about meningitis as an extrahepatic manifestation of hepatitis C is very limited [12, 27].

HCV infection is an independent predictor of stroke and cerebrovascular death [28]. HCV viral load is independently associated with early, asymptomatic carotid atherosclerosis [29]. However, there are only single reports of incidence of acute aseptic meningitis as an extrahepatic manifestation of hepatitis C [12, 27].

Viral meningitis is the most common form of meningitis worldwide [30]. Viral infection causes the inflammation of the meninges with an associated abnormal cell count in the cerebrospinal fluid [31]. Appropriate and timely evaluation is critical. As in the above-described clinical case, meningitis typically presents with the acute onset of fever, headache, photophobia, neck stiffness, nausea, vomiting, and signs of meningeal irritation [32]. Enteroviruses (Coxsackie or Echovirus groups) are the most common cause of viral meningitis across all age groups [33]. Herpes viruses that cause meningitis include herpes simplex virus 1 and 2, varicella-zoster virus, cytomegalovirus, Epstein-Barr virus, and human herpesvirus 6. Other viral causes include adenovirus, lymphocytic choriomeningitis virus, influenza, parainfluenza, and mumps [33]. Arboviruses that can cause viral meningitis include West Nile virus, Zika, chikungu-

ORCID and contributionship:
Olena Y. Fartushna: 0000-0002-4641-0836 A,B,D,F
Maria M. Prokopiv: 0000-0001-5467-3946 A,D,F
Victoria Y. Krylova: 0000-0002-3029-3663 E,F
Svitlana V. Rohoza: 0000-0002-4867-0340 E,F
Hanna V. Palahuta: 0000-0001-7348-4390 A,B,E,F
Yana Y. Hnepa: 0000-0002-0276-1466 D,E,F
Yevhen M. Fartushnyy: 0000-0002-5199-5373 D,F

Conflict of interest:
The Authors declare no conflict of interest.

CORRESPONDING AUTHOR
Olena Ye. Fartushna
Ukrainian Military Medical Academy
24 Melnikova St., 04050 Kyiv, Ukraine
tel: +380 (97) 7911003
e-mail: olena.y.fartushna@gmail.com

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