INTRODUCTION
Herpes zoster (HZ) is an often painful disease caused by varicella-zoster virus (VZV) infection. Zoster affects approximately 1,000,000 individuals in the U.S. per year. Most patients are over age 60 [1] or immunocompromised [2]. The annual incidence of zoster is 5 to 6.5 per 1,000 patients at age 60, who visit hospitals each year, increasing to 8 to 11 per 1,000 at age 70 [2]. Unlike varicella, which occurs primarily in the spring, there is no seasonal predilection for zoster. Patients with HZ complain of painful, vesicular rashes with erythema, which usually take 3~4 weeks to heal [3].

Varicella zoster virus (VZV) is an ubiquitous, exclusively human neurotropic \textit{alpha}herpesvirus that causes the predominantly childhood disease chickenpox (varicella) during primary infection of susceptible individuals. After resolution of primary infection by the host immune system, the virus can establish a life-long, latent infection in cranial nerve ganglia, dorsal root ganglia and autonomic ganglia along the entire neuraxis. At some stage later in life, as cell-mediated immunity to VZV declines with age or immunosuppression latent VZV eventually-reactivates, presumably in a single sensory neuron, to cause herpes zoster (shingles). The reactivated virus multiplies and spreads within the ganglion, infecting many additional neurons and supporting cells—a process that causes intense inflammation and neuronal necrosis, often followed by chronic pain (postherpetic neuralgia or PHN) as well as myelopathy, encephalitis, meningitis, Ramsay-hunt syndrome with facial paralysis, retinal necrosis and cerebellitis [4-7]. VZV is able to replicate in the walls of cerebral arteries, causing vasculopathy and VZV-associated small vessel disease [8, 9]. Due to macrovascular disease, granulomatous angiitis often develops, which leads to stroke [10]. VZV-associated small vessel disease has such manifestations as migraine, convulsions, paralysis, and cognitive impairment. HZ is predominantly a marker of immunodeficiency [11]. VZV reactivation can also produce pain without rash (zoster sine herpete). In fact, all neurological complications of VZV reactivation can occur without rash. More than 90% of adults in the World have serologic evidence of prior VZV infection. Consequently, latent VZV is present in the sensory ganglia of virtually every older adult. Thus, almost every older adult in the Ukraine is at risk of developing herpes zoster. In recent years, an increase in the number of patients with herpes zoster is noted not only among the elderly, but also a shift in diseases towards the middle and younger age [8]. Because VZV becomes latent in ganglia along the entire neuraxis, zoster can develop anywhere on the body. Zoster can affect all cranial nerves [12] and...
spinal nerves at all levels; zoster can also be associated with lower motor neuron type weakness in the arm or leg [13], diaphragm [14] or abdominal muscles [15].

THE AIM
To explore the clinical features, diagnosis, and treatment of CNS injury caused by VZV infection in a prospective single center study from January 2014 to January 2018.

MATERIALS AND METHODS
117 patients aged 18 to 74 years old, meeting the initial criteria of CNS disorders caused by VZV were enrolled in the study. VZV-etiology of the nervous system lesions was confirmed by the presence in patients of a typical exanthema, clinical neurological analysis, neuroimaging, as well as the identification of VZV infection markers (viral DNA and IgM antibodies). Among the examined patients, 62 (53.4%) were female and 55 (46.5%) males. Young people were predominant.

All experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the Declaration of Helsinki.

RESULTS AND DISCUSSION
The main clinical manifestations of the VZV neuroinfection were ganglionitis and ganglioradiculoneuritis - in 81 (69.8%) patients. Cranial ganglioneuritis were detected in 36 (31.0%). Most commonly affects Gasser's ganglion – in 28 (24.0%) patients, less commonly - cranial nodes - 8 (6.8%). Isolated lesion of the first branch of the trigeminal nerve was observed in 21 patients, the second - in 5 one, the third - in 2 one. HZ is commonly associated with severe diffuse headache, and unilateral pain, which often precedes the rash by several days; pain usually accompanies the dermatomal rash of herpes zoster. In 9 patients with a defeat of the first branch of the trigeminal nerve the conjunctivitis on the side of the lesion developed, in 2 – keratitis, and one – uveitis. The defeat of the second and third branches of the trigeminal nerve was accompanied by the appearance of a rash on the mucous palate, inner surface of the cheeks, and lips. In 3 patients, paresis of the eye muscles was observed. Herpes zoster is virtually impossible to diagnose until the characteristic vesicular dermatomal rash appears.

In 8 patients with lesions of the cranial nerve and the tympanic strike - Ramsey-Hunt's syndrome were diagnosed. Vestibular, trigeminal, facial and sublingual nerves were also involved in the process. The rashes were localized on the skin of the auricle, external auditory canal, anterior surface of the tongue, posterior surface of the palate. Vestibular disorders, partial loss of hearing, peripheral paresis of mimic muscles accompanied with pain of neuralgic in nature, paresis of soft palate and disturbance in swallowing were observed.

Spinal ganglia were involved in 45 (38.7%) patients. Lower cervical and upper chest regions were implicated in 9 of them, chest - in 30, and lumbar sacral – in 6 patients. In the clinical picture of ganglionitis of the lower cervical and upper chest localization burning pain in the hand, sensation of swelling of the brush, muscles hand paresis dominated. Shingles pains, and paresthesia were noted in all patients with lesions of the thoracic ganglia. Weakness of the abdominal wall, decreased muscle tone, imitate neoplasia formation in the abdominal cavity of 3 patients. In 26 patients there was a defeat of several sensitive nodes. Spastic paresis, and central type function disturbance of the pelvic organs developed in 4 patients.

Ganglionitis of the lumbosacral region are characterized by the rash localized on the skin of the lumbar, buttocks, lower extremities, accompanied by significant pain syndrome, symptoms of the tension of the spinal nerve roots, weakness of the lower extremities, violation of the urination and defecation, which was considered as myelaganglionitis.

Meningoencephalitis has developed in 6 patients, in 3 - on the underlying defeat of the Gasserov's node, and 3 - in the defeat of the thoracic spin ganglia. In 2 cases, VZV-encephalitis was not accompanied by a defeat of other parts of the nervous system. Common symptoms were observed in all patients; meningeal contractures were moderate in 11 patients. Lymphocytic pleocytosis was found in the liquor. Meningitis in 13 patients accompanied with rashes in some spinal dermatomes. In one 18 years old patient with injury of the first branch of the Gasserov's node on day 7 after the onset of the disease contralateral hemiparesis developed. Ultrasound analysis of intracranial vessels, had shown a reduced blood flow in the system of the middle cerebral artery on the side of herpetic defeat, and signs of venous discirculation. The event was considered as the onset of cerebral angitis.

The participation of VZV in lesions of the nervous system is hard to detect and prove hard to prove when there are neurological symptoms but no typical exanthema and indication on HZ in the history. 10 (8.6%) patients, have radicular pains in the chest dermatomas without anamnestic HZ. In 5 (4.3%) patients with cephalic syndrome, cognitive impairments, multiple small subcortical foci in the hemisphere white matter VZV-vasculopathy was diagnosed, although there was no herpes history. CSF samples analysis revealed specific antibodies, and VZV DNA.

CASE PRESENTATION
A 34-years-old man, invalid of the III group, presented to The Center of Infectious Disorders of the Nervous System (CIDNS, Kyiv, Ukraine) in 07.09.2017 with complaints of a sharp weakness in the left limbs, left side partial loss of vision and hearing, double vision, headaches, weakness, dizziness, decreased ability to work and coordination, subfebrile. It is known from the medical history that he is ill from the middle of August 2017, when being treated in a local hospital for ketoacidosis, acute left-sided weakness and problems with vision and hearing suddenly appeared. He underwent MRI (figure 1) which showed a massive

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formation in the cerebellum, possibly a tumor. After consulting a neurologist and a surgeon, the patient was referred to a CIDNS with suggestive diagnosis focal brain damage, probably infectious or malignant genesis. History of life: it is afflicted with chronic gastroduodenitis, spinal osteochondrosis, insulin-dependent diabetes mellitus (from 2013), stage of decompensation, ketoacidosis. In 2013, laparotomy with resection of pancreatic cysts was performed. He is alcohol abuser. He suffered chickenpox in childhood.

On admission: general condition of moderate severity. The emotionally labile, contact, answer the question adequately. Skin and mucous membranes of normal color, mucous pharynx is hyperemic. Injection of vessels of sclera. Pupils are the same, photoreaction is not reduced. Eyelid tremor with ptosis of the left eyelid, and not completed convergence, more right, are observed. There are left, horizontal nystagmus, smoothness of the left nasolabial fold, deviation of the tongue and uvula to the left. Muscular strength is reduced on the left limbs (hand to 3 points, foot up to 2 points). Abdominal reflexes are absent. Tendon reflexes on the upper limbs saved, S> D, on the lower extremities S> D. Barre-probe as well as Gordon, Stryumpel Sharapov, Babynsky symptoms were positive on the left side. Meningeal symptoms were not detected. He performed the coordinate tests with light intent. In the Romberg pose he fell to the left and back. The function of pelvic organs was not disturbed.

On admission, the blood analysis had shown normocytosis (5 x 10^9 / l) with relative lymphocytosis (50%), increase gamaglutamyltransferase (59.8U/l), and hyperglycemia (15.9 mmol / l). IgM antibodies to VZV and elevated levels of autoantibodies to neuroantigens were also detected (BMP of 42.8 U / ml, S-100 of 13.9 U / ml, NSE of 32.8 Un / ml, GHMA of 37 Un / ml). All CSF indicators were within normal range. Microflora, herpesvirus DNA, toxoplasma and mycobacterium tuberculosis have not been detected. The presumptive diagnosis was: encephalitis with lesion of cerebellar and brain structures, pronounced vestibular atactic, cerebrospinal symptoms, left-side hemiparesis, polyneuropathy, underlying with VZV infection in the phase of reactivation. Diabetes mellitus type 1, severe course, stage of decompensation. Chronic gastroduodenitis. Osteochondrosis of the lumbar spine.

He was treated with iv acyclovir, 1500 mg / per day; human immunoglobulin for iv administration, neuroprotective, hepatoprotective, and anti-inflammatory therapy. As a result of treatment, stay of the patient improved: increased muscle strength, vision and hearing normalized, polyneuropathy, and ataxia almost disappeared, glucose blood level normalized. IgM VZV antibodies in the blood test of September 20, 2017, were not detected. The patient was discharged for further outpatient treatment. Three months after discharge, the control test of CSF sample revealed not VZV DNA nor antibodies against virus. The level of autoantibodies to neuroantigens became almost normal.

CONCLUSIONS

The most common clinical neurological variations of HZ were ganglionitis (69.7%) had cranial localization in 31% of patients with spinal - 38.7%, the defeat of the meninges - the 16.3% lumbar puncture should be conducted in patients with herpes zoster, especially with lesion of cranial ganglia. Lack typical exanthema not preclude nervous system VZV and should use targeted methods etiology decoding process, cerebrospinal fluid testing for DNA specific antibodies.
REFERENCES


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

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