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CASE REPORT

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Facial herpes zoster complicated by cerebral oedema in the course of encephalitis

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ABSTRACT

Herpes zoster is the result of the reactivation of the varicella-zoster virus (VZV), which remains dormant in the sensory ganglia. The clinical presentation is characterized by a unilateral vesicular rash occurring on an erythematous background and most commonly affecting one dermatome of the skin. An extremely rare complication of herpes zoster is encephalitis, which occurs in approximately 0.2% of cases. It involves fever, headache, nausea, vomiting, impaired consciousness, hallucinations, and balance disturbances in addition to the rash. The study presents a case of a 71-year-old female patient hospitalized in the Dermatology Clinic due to zoster on the left side of her face, accompanied by the above symptoms that occurred 2 days before admission and brain oedema observed in the head computed tomography, constituting the clinical picture of VZV-related encephalitis.

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Keywords: herpes zoster, varicella-zoster virus, encephalitis, cerebral oedema

INTRODUCTION

Herpes zoster accounts for 1% of infectious skin diseases. The etiological agent is varicella zoster virus (VZV), a pathogenic human herpes virus that causes varicella (chickenpox) as a primary infection and becomes latent in the peripheral ganglia. Varicella zoster virus reactivates either spontaneously or due to several triggering factors, involving reduced immunity, to cause herpes zoster (shingles). The clinical picture of a zoster is a unilateral, small vesicular erythematous skin lesion, corresponding to one or more skin dermatoses. The lesions can burst easily and leave scars. Most often, the skin lesions do not cross the midline of the body. The lesions may be accompanied by tingling, burning, itching or significant pain in the dermatomes. In 70–80% of cases, these symptoms precede the onset of skin lesions by 3–4 days. The most common complication of herpes zoster is postherpetic neuralgia with a benign course, which reduces the quality of the patient's life. Other neurological complications of herpes zoster include central nervous system (CNS) pathologies, including cerebellar ataxia, cerebral arteritis, myelitis, meningitis and encephalitis. Central nervous system infection can occur in the course of primary or secondary reactivation of VZV [1].

CASE REPORT

A 71-year-old woman presented to the Dermatology Department due to left side facial zoster. The first skin lesions appeared 2 days before hospitalization and were preceded by a severe headache, nausea and vomiting, which had occurred 4 days earlier. The patient began treatment with acyclovir on an outpatient basis but was unable to take the prescribed medication due to severe vomiting. Symptoms on admission to the Dermatology Department involved numerous small vesicles on an erythematous background, crust and erosions on the skin of the forehead, temple, and parietal area on the right side, and swelling of the left eyelid (Fig. 1). No other abnormalities and meningeal symptoms were found. Laboratory tests revealed anti-VZV antibodies in both IgM and IgG classes. Due to persistent vomiting and headaches responding poorly to analgesics, a head computed tomography (CT) scan without contrast was performed, which revealed cerebral oedema (Fig. 2). As

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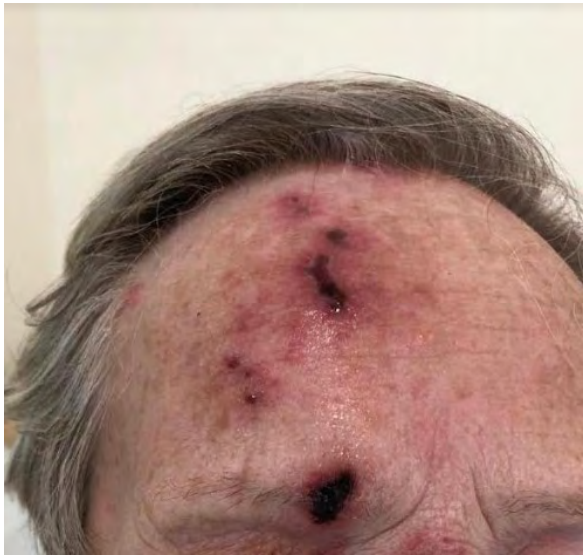

Figure 1. Numerous small vesicles on an erythematous background, crust and erosions on the skin of the forehead, temple, and parietal area on the right side

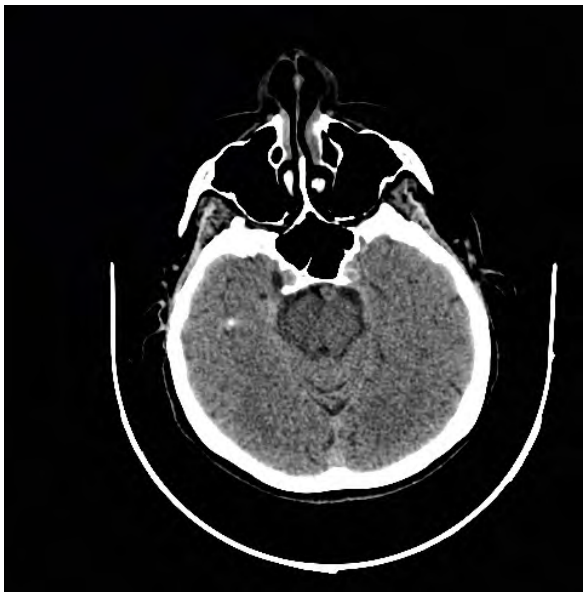

Figure 2. Rubbing of the furrows in both hemispheres of the brain especially in the temporal lobes, occipital lobes and subthalamotically

a result a lumbar puncture for further diagnosis was abandoned. Based on the clinical picture and additional tests, a diagnosis of VZV encephalitis was made and intravenous acyclovir at the dose of 500 mg 3 times daily was administered. Symptomatic treatment of cerebral oedema involved a parental dosage of 100 mL 20% mannitol with 40 mg of furosemide, dexamethasone (16 mg — 8 mg — 0 mg *i.v.*) and metoclopramide. The pain and nausea resolved on the day of the anti-oedema therapy. During hospitalization in the Dermatology Department, the patient reported an increasing cough, without dyspnoea. In laboratory tests SARS-CoV-2 antigen was positive. The patient was transferred to the Department of Infectious Diseases hospitalizing patients with COVID-19 for further treatment. Over the next few days, the patient's vital signs deteriorated, and respiratory and multi-organ failure worsened, which unfortunately led to the patient's death in the Intensive Care Unit.

DISCUSSION

Varicella zoster virus is the second most common cause of viral encephalitis and viral meningitis, affecting only 0.1––0.25% [2, 3] of patients with zoster. It is more common in disseminated cases and *foci* involving dermatomes in close proximity to the vicinity of the central nervous system. Two main risk factors for developing the zoster are the impaired immune system and the age of patients over 65. This is particularly crucial in cases of potential virus transmission from grandchildren, as previously described [4]. In a retrospective analysis, Skripuletz et al. [3] reported an isolated vesicular rash as the most common manifestation among 282 patients diagnosed with zoster and hospitalized at the Department of Neurology in Hannover. In half of those patients, it originated from the trigeminal ganglion and 90% of these cases involved an ocular nerve as the first branch of the trigeminal nerve. In 21% of patients, it initiated in the spinal ganglia and almost half of them had involved dermatomes of the thoracic spinal nerve. In the same study, the nervous system was affected in 12% of patients with zoster. The mortality rate of VZV encephalitis in immunocompetent patients is about 15% and almost 100% in immunosuppressed patients, especially in cases of additional liver or lung involvement. This group of patients requires special attention, and VZV encephalitis must be considered in cases of both drug- and disease-induced immunosuppression [5, 6]. Clinical signs of VZV encephalitis are headache, fever, nausea, vomiting, disturbance of consciousness, and productive symptoms such as hallucinations or convulsions. These symptoms can often be misdiagnosed as side effects of valacyclovir used in zoster therapy, especially in patients with impaired renal function. The rash may precede or occur after the onset of neurological symptoms. As for the present patient, the rash appeared 3 days after the headache, nausea and vomiting. Neurological symptoms accompanying skin lesions and hemiplegia oblige us to perform brain imaging studies — non-contrast CT or magnetic resonance imaging (MRI). They can show reduced hypodensity of the temporal lobes, with possible involvement of the frontal lobe, sparing of the basal nuclei (in CT) or features of oedema with excessive density in the mentioned locations and sparing of the brain base (in MRI). The gold standard for diagnosing VZV encephalitis is a lumbar puncture and examination of cerebrospinal fluid. This procedure can detect signs of viral

infection, characterized by clear fluid, a slightly increased number of cells, and normal or slightly decreased glucose levels [4, 5, 7]. It also allows for the identification of viral DNA or anti-VZV antibodies, leading to a correct diagnosis [5, 6]. However, it should be remembered that lumbar puncture is contraindicated in the case of cerebral oedema, and any features of cerebral oedema should be excluded by CT scan or ophthalmoscopic examination. Due to the features of cerebral oedema on CT in the study patient, lumbar puncture was abandoned. The treatment of choice for zoster with encephalitis remains intravenous acyclovir. Many authors have noted the clinical efficacy (defined as reduced mortality or neurological complications) of acyclovir and ganciclovir in the treatment of VZV encephalitis in HIV-infected [8], immunocompromised [7, 9–11] and immunocompetent patients [12–14]. Broucker et al. [11] and other authors [15, 16] have not observed a significant impact of different doses of acyclovir (from 10 to 15 mg/kg/8 h) and time of therapy (from 14 to 21 days) on the clinical outcome in patients. Due to a lack of other clinical evidence for the efficacy of a higher dose of acyclovir and the optimal length of therapy, the majority of recommendations are 15 mg/kg body weight/8 h for 10 to 14 days and prolonging the duration of therapy in immunocompromised patients. In the present case, additional symptomatic treatment of cerebral oedema was implemented.

CONCLUSIONS

Clinical observations suggest that in the case of vomiting and severe headaches non-responding to treatment, one should consider the possibility of neurological complications of a common infectious disease such as varicella zoster. In such a case, diagnostic imaging tests (CT and/or MRI) should be performed. The length of intravenous acyclovir therapy for VZV encephalitis has not been precisely established. The effective dose of acyclovir is 10–15 mg/kg/8 h and the optimal duration of therapy is from 10 to 21 days. There is an increased risk of neurological complications in the course of VZV infection in elderly patients, burdened with additional diseases. Early diagnosis and inclusion of appropriate treatment significantly reduce the risk of death. The case presented here is intended to sensitize doctors to the rare neurological complications in the course of a zoster.

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Author contributions

Data analysis and preparation of the manuscript — JC, AS, JC; work plan and the evaluation of the content — NB; JC, JN, AL.

Conflict of interest

The authors declare no conflict of interest.

Ethics statement

No ethical issues.

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