Chorioretinitis Associated with HSV-1 Neuroinfection Following Viral Infection. A Case Report

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SUMMARY

Introduction: Chorioretinitis is an inflammatory disorder affecting the choroid and the retina, classified among posterior uveitis conditions. It is clinically manifested in central visual impairment, visual field defects, and in more severe cases may lead to irreversible loss of vision. Accurate differentiation between infectious and non-infectious etiologies is essential, as treatment strategies differ significantly. The prognosis depends on early diagnosis and timely initiation of targeted therapy.
Purpose: To present a case of a rare form of chorioretinitis associated with HSV-1 reactivation following a recent parvovirus B19 infection.
Case presentation: A 35-year-old female patient reported to our center in June 2024 with acute deterioration of vision in her left eye. Her medical history revealed that approximately 10 days prior to the examination, the patient had suffered a viral illness with exanthematous manifestations, diagnosed as parvovirus B19 infection, which was managed symptomatically. A fundoscopic examination revealed inflammatory changes in the macular region. As part of differential diagnostics, fluorescein angiography, OCT, perimetry, serological testing and lumbar puncture were performed. A molecular analysis of cerebrospinal fluid confirmed HSV-1 by PCR. Antiviral treatment with acyclovir (750 mg i.v. every 8 hours, followed by 400 mg orally twice daily for 6 weeks) led to clinical stabilization and improvement of central visual acuity to 20/25.
Conclusion: The presumed association between parvovirus B19 infection and subsequent HSV-1 reactivation indicates a potential immunomodulatory effect. This case underscores the importance of a multidisciplinary approach and prompt therapeutic intervention in the case of viral neuro-ophthalmic complications.
Key words: posterior uveitis, OCT, HSV-1, parvovirus B19, neuroinfection

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INTRODUCTION

Chorioretinitis is classified as a type of posterior uveitis affecting the choroid and the retina [1]. The choroid has an abundance of blood vessels and plays a key role in supplying the outer layers of the retina with nutrients and oxygen. Consequently, inflammation in this region can cause severe complications, which seriously endanger the patient's sight. This inflammation frequently affects not only the choroid but also the adjacent structures, especially the retina, potentially impairing its function and structure. This damage may lead to loss of visual acuity, the appearance of scotomas and other defects of the visual field, which often causes the patient to seek medical attention from an ophthalmologist. It is important to ensure timely detection of the symptoms and a quick therapeutic solution in order to avert permanent damage to sight [2]. The diagnosis of chorioretinitis requires a comprehensive and precise approach, which is essential for identifying the etiology and ensuring that the correct therapeutic decision is made [3]. Ophthalmological examination represents the foundation of diagnosis, and incorporates an evaluation of visual acuity, analysis of the visual field and a detailed examination of the ocular fundus with the aid of ophthalmoscopy [4]. Modern imaging techniques are used in order to confirm affliction of the choroid and retina, in particular fluorescein angiography (FAG) and optical coherence tomography (OCT). Fluorescein angiography enables visualization of vascular abnormalities and impaired permeability, whereas OCT provides layered high-resolution images of the retina, thereby highlighting structural changes in detail [5,6]. An important element of the diagnostic process is laboratory tests, which help identify systemic disorders and infectious agents. Serological tests detect antibodies against various micro-organisms, PCR analyses enable precise identification of pathogens, and immunological examinations indicate the presence of autoimmune mechanisms [7–9]. The

therapeutic approach is individualized and depends on the etiology [2,10]. Successful diagnosis and treatment of chorioretinitis requires a multidisciplinary approach. Cooperation of ophthalmologists, immunologists and other specialists is essential in order to ensure comprehensive patient management, which minimizes the risk of complications and supports the preservation of visual functions. This systematic approach emphasizes the importance of timely detection and treatment, which are crucial for optimizing the clinical results.

CASE REPORT

A 35-year-old female patient was admitted at our ophthalmology clinic at the beginning of summer 2024 with an acute deterioration of visual acuity in the left eye, which had persisted for approximately two days. Her personal medical history revealed that she had recently recovered from fifth disease, caused by the B19 parvovirus and characterized by a typical rash on the arms and legs, which was treated symptomatically and had appeared approximately 10 days before our examination. At the same time the patient stated that she was suffering headaches and dizziness, which had appeared in the last two days and preceded the deterioration of her vision. These symptoms indicated a possible systemic or neurological connection with her current ophthalmological condition. The patient did not state any other systemic symptoms such as fever, myalgia or arthralgia. Her subjectively perceived deterioration of visual functions and associated neurological symptoms required an immediate and thorough examination. The symptoms indicated an inflammatory process affecting the posterior segment of the eye, which may have been associated with a previous viral infection or other severe systemic complication. Due to the suspicion of an inflammatory disorder affecting the posterior segment of the eye, we continued to examine the patient further with the aid of advanced imaging techniques and laboratory analyses.

At the initial ophthalmological examination, a pronounced deterioration of central visual acuity was recorded in the left eye with a value of 6/36, whereas normal visual parameters were recorded in the right eye. The patient stated subjective blurred vision and inability to focus on objects in the central area of the visual field, as well as a dark gray band with flecks and mist in the surrounding area. The clinical picture corresponded to an active inflammatory process. Upon examination on a slit lamp, infiltration with presence of multiple small dotted intraretinal hemorrhages was observed in the region between the superior and inferior temporal arcades (Figure 1). Neither the anterior nor the posterior segment of the eye manifested any other signs of inflammatory activity. These findings were highly suggestive of chorioretinitis, characterized by typical manifestations of acute inflammation localized in the macular region.

An OCT examination was conducted in order to ensure a more precise evaluation of the scope of affliction (Figure 2). The examination confirmed damage to the macular region with the presence of subretinal fluid, multiple cystic formations and impairment of the continuity of the outer photoreceptor layer. These findings attest to edema and an actively ongoing inflammatory process which requires targeted and timely therapeutic intervention. Fluorescein angiography (FAG) was conducted to supplement the diagnosis (Figure 3), which enabled us to assess the dynamics of vascular changes in the retina and the presence of signs of active inflammation. The detailed phases of the examination and the corresponding findings are presented in the key to the image.

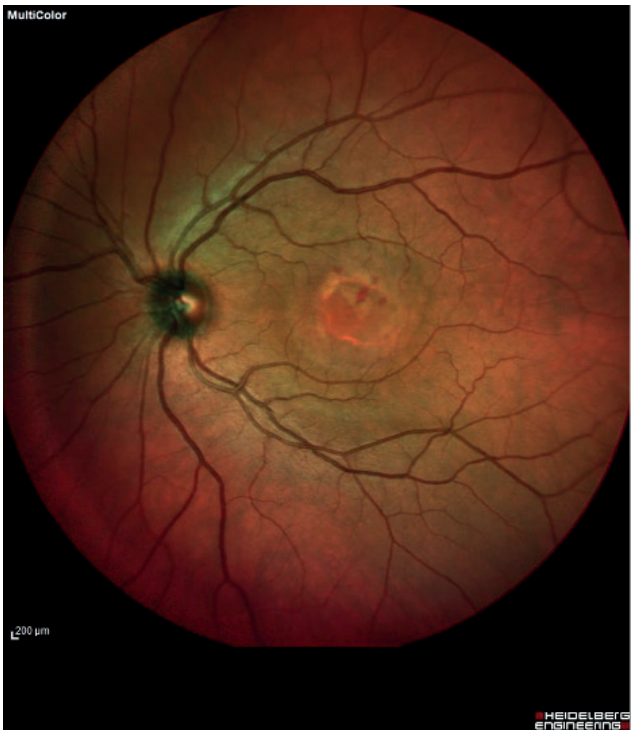

Figure 1. Fundoscopic image during the patient's initial examination

A perimetric examination detected the presence of partial central scotoma in the left eye – reduced blind spot in the central part of the visual field, which may be the consequence of an inflammatory process or edema in the macular region. This finding was in accordance with the clinical picture of chorioretinitis (Figure 4).

Based on the combination of clinical symptoms, imaging methods (OCT, FAG) and functional perimetric examination, the patient was diagnosed with suspected infectious form of chorioretinitis, requiring further diagnostic and therapeutic intervention.

With regard to the combination of ophthalmological symptoms, pronounced deterioration of visual acuity, central scotoma, macular changes on the OCT image, as well as the presence of hyperfluorescence in the macular region during FAG examination together with the associated systemic symptoms such as headaches and dizziness, possible neuroinfection was suspected. For this reason, the patient was subjected to a neurological examination

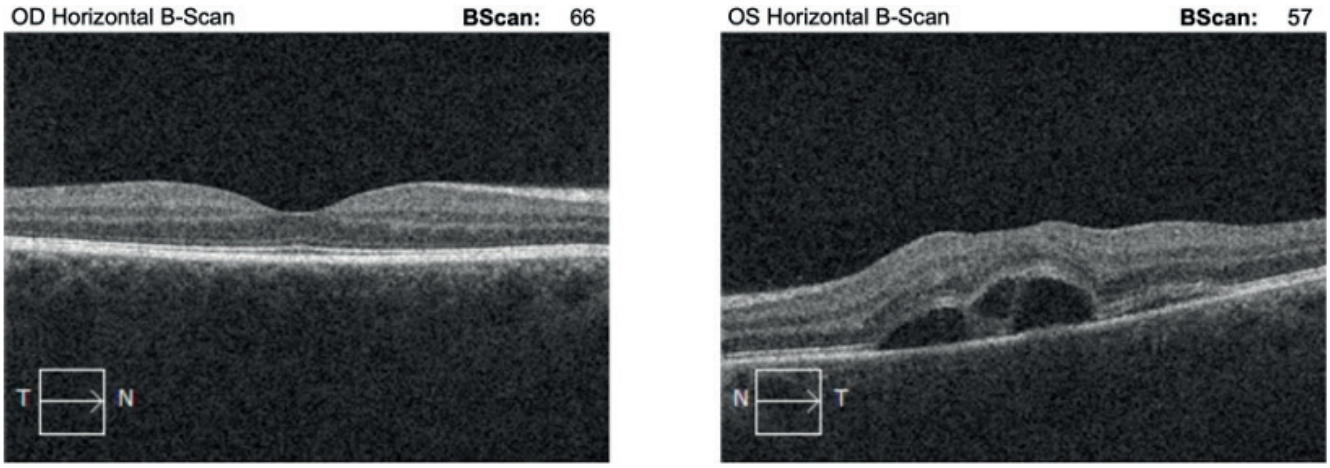

Figure 2. Optical coherence tomography of the left eye showing significant pathological changes, and of the right eye with a physiological finding

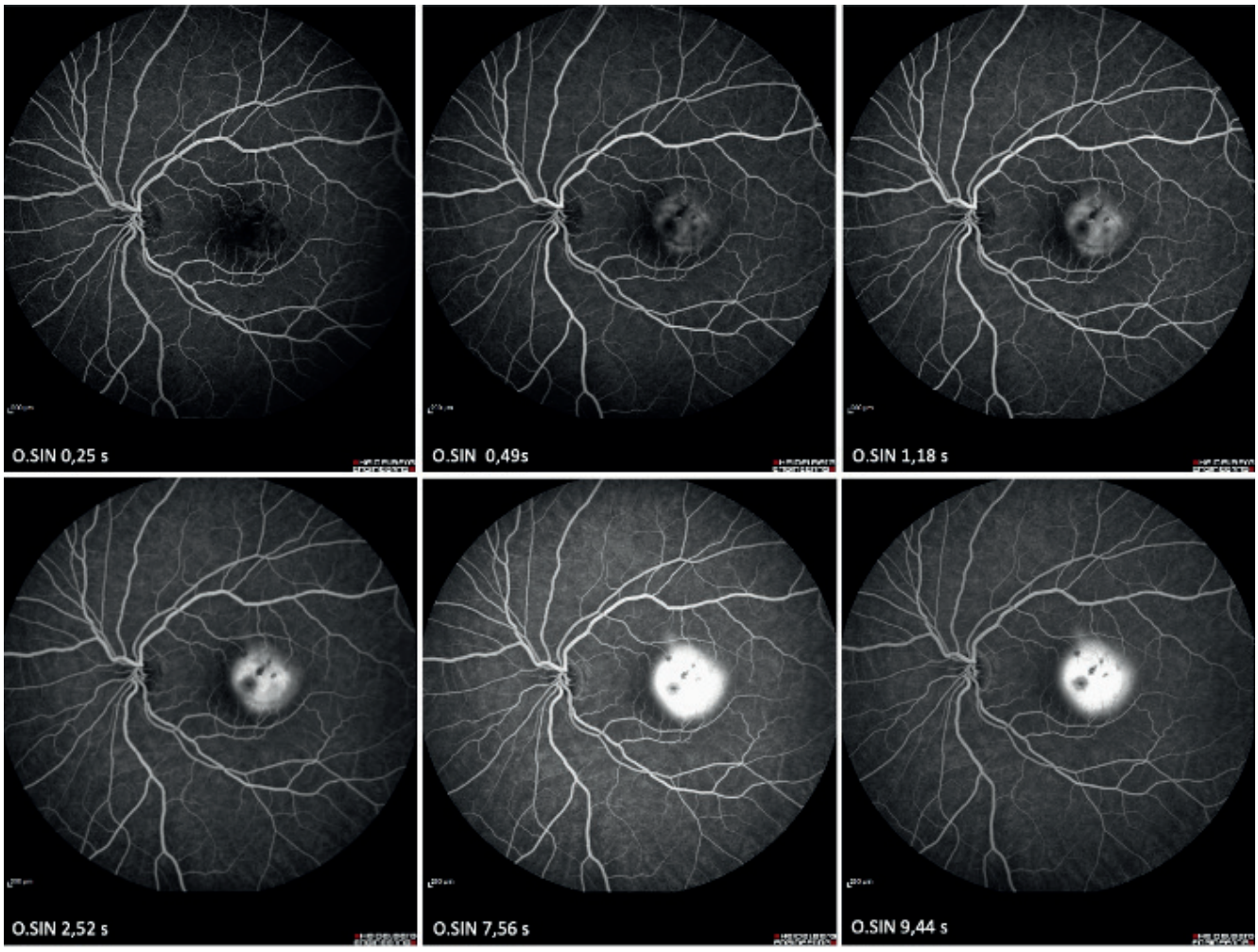

Figure 3. Series of fluorescein angiography (FA) images of the left eye, showing various phases of retinal vessel perfusion and inflammatory changes in the macular region. The top row presents the early phases of FA, demonstrating rapid filling of the retinal vessels with fluorescein. A hyperfluorescent lesion is visible in the macular area, possibly caused by oedema in this region. The bottom row depicts the late phases of FA, revealing hyperfluorescence in the macular area, indicating fluorescein leakage due to increased vascular permeability and inflammatory process. A blurred margin of the hyperfluorescent lesion, confirming active inflammation, is also typical

including magnetic resonance. The neurological examination did not detect any motoric or sensory deficits, the patient had no meningeal symptoms, and magnetic resonance imaging did not indicate a demyelization process, the image showed no presence of acute or subacute malacia of brain tissues. However, the confirmation of central

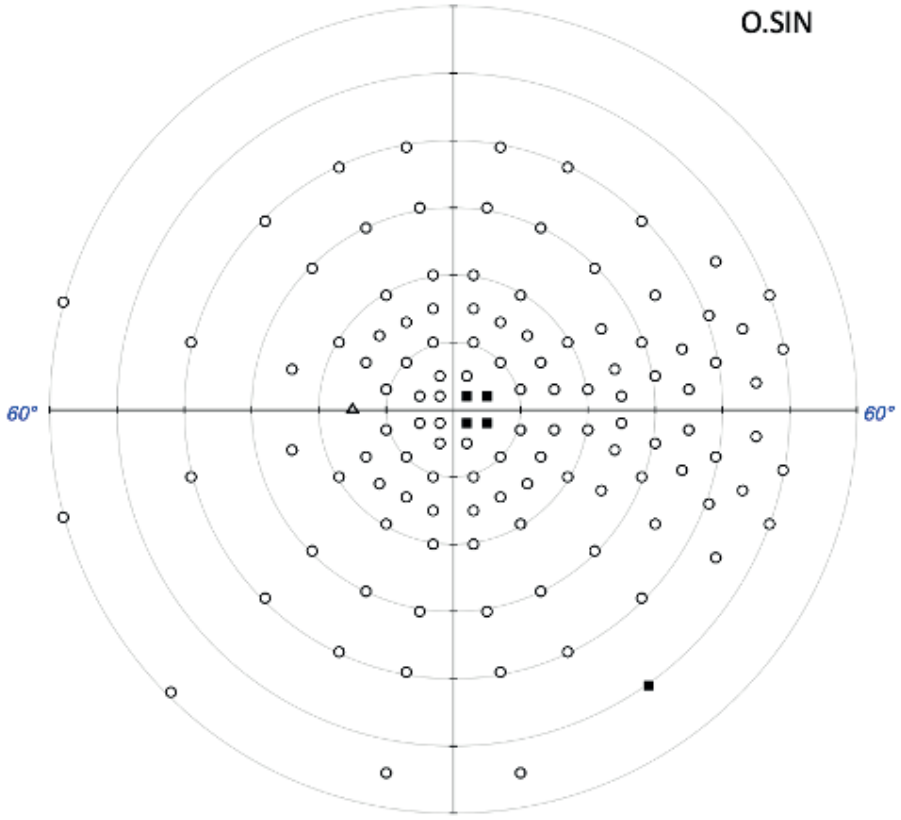

Figure 4. Partial central scotoma detected on perimetric examination

scotoma by perimetric testing indicated a possible inflammatory process influencing the central nerve structures associated with the visual pathway.

A series of laboratory tests were performed in order to clarify the etiology. Lumbar puncture was performed as part of differential diagnostics, with the aid of which the cerebrospinal fluid (CSF) was sampled for biochemical and serological analysis. The CSF was clear, with slight pleocytosis, which indicated an inflammatory process. At the same time serological samples were taken from the serum, which incorporated tests for a broad spectrum of infectious agents such as Parvovirus B19, Rubeola, Toxoplasma gondii, Borrelia bundorferi, Treponemma pallidum, HIV and herpes viruses, including herpes simplex virus type 1 (HSV-1). The serological tests demonstrated an absence of active infection of the examined agents, thereby excluding other possible infectious etiologies, but the PCR test for Parvovirus B19 showed a weak positive, which merely confirmed that the patient had recovered from fifth disease.

The key result was the polymerase chain reaction (PCR) from the cerebrospinal fluid, which detected the presence of HSV-1 DNA, thereby definitively confirming a diagnosis of neuroinfection caused by herpes simplex virus type 1 (HSV-1). Immediately after the determination of diagnosis of HSV-1 neuroinfection with chorioretinitis, the patient was administered targeted antiviral therapy. With regard to the acute phase of the pathology and the severity of the clinical symptoms, after consultation with the Department of Infectology and Geographical Medicine at the Faculty of Medicine of Comenius University, intravenous therapy with acyclovir in a dose of 750 mg every 8 hours over a period of 14 days was indicated. Acyclovir, as a nucleoside analogue with a specific effect against the herpes simplex virus, was the therapy of first choice, since it has the capacity to inhibit viral DNA polymerase and thereby halt replication of the virus. Intravenous application was preferred in order to guarantee sufficient therapeutic concentrations in the affected tissues, including the ocular structures and the central nervous system.

After discharge from hospital and an improvement of her acute condition, the patient continued with oral maintenance therapy with acyclovir in a dose of 400 mg per day for a period of 6 weeks. This long-term therapeutic regimen was chosen in order to suppress the replication of the residual virus and to prevent relapses, while oral administration ensured appropriate compliance on the part of the patient. During the course of treatment a gradual improvement of the patient's clinical condition was observed, including subsidence of the inflammatory changes on the retina. This development was systematically monitored with the aid of ophthalmoscopy and repeated OCT, which confirmed a positive therapeutic response (Figure 5). Images 5A and 5B show the findings from the first days after the commencement of treatment. The fundoscopic image (5A) indicates a persistent central macular lesion with residual signs of inflammation. The OCT examination (5B) detected the presence of macular edema, thickening

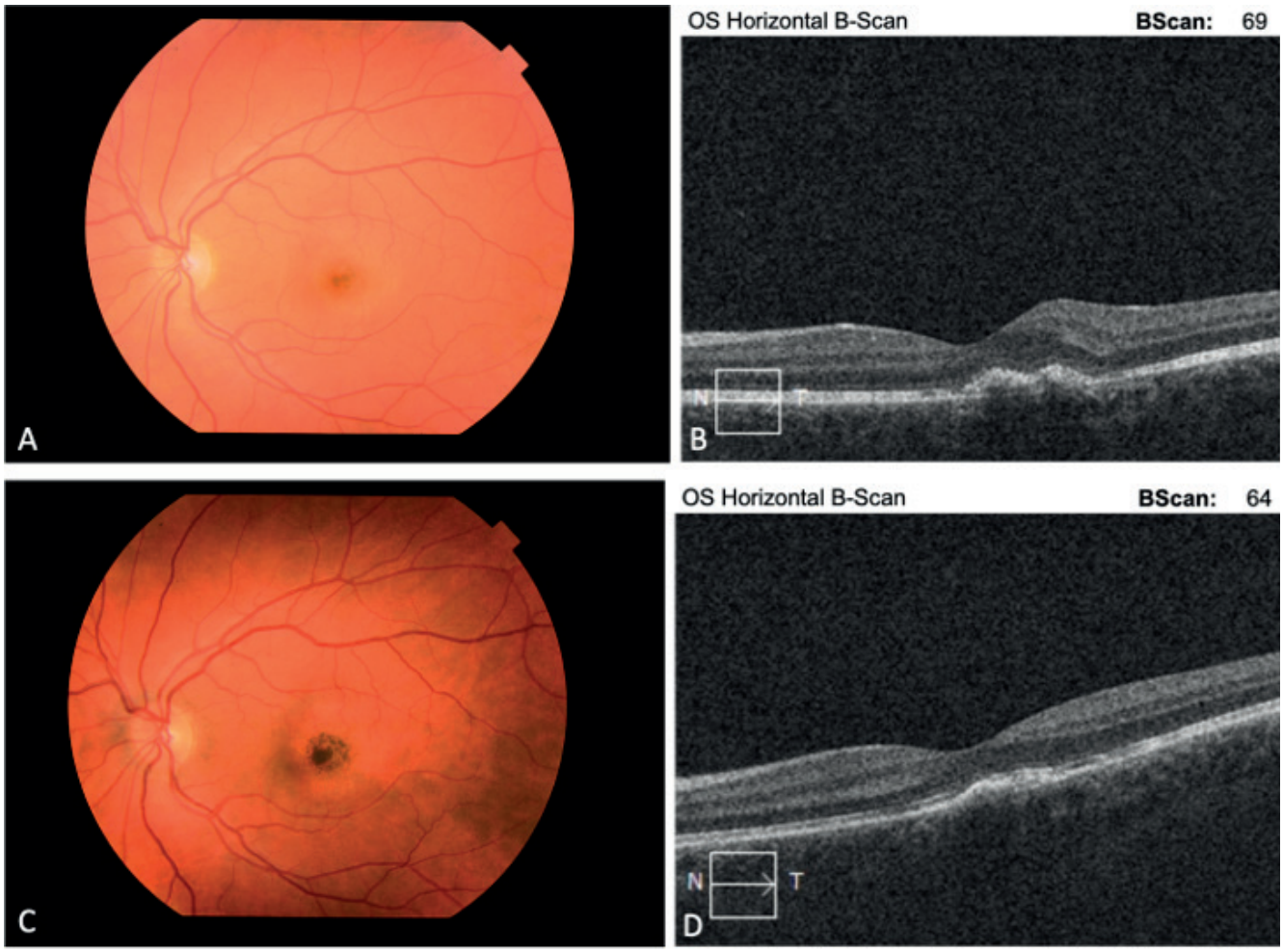

Figure 5. Images documenting disease progression and treatment efficacy

and impairment of the layering of the retina, which was consistent with a persistent active inflammatory process. After the continuation of treatment, images 5C and 5D document stabilization of the condition. Fundoscopy (5C) demonstrated the regression phase of the inflammation – the macular lesion was less active, with accentuation of pigmentation. The OCT image (5D) showed a significant reduction of edema, restoration of the regular architecture of the retinal layers and a reduction of thickening, which attested to a positive response to treatment.

In order to support the evaluation of anatomical changes, 3D optical coherence tomography was also added (Figure 6), which provides a detailed image of the differences before and after treatment. The 3D OCT scan before treatment (6A) shows pronounced structural impairment of the macular region with edema, increased retinal thickness and irregularities in the outer layers – a finding typical of active inflammation. The follow-up scan after treatment (6B) shows reduction of edema, restoration of the regular layering of the retina and a return to a physiological anatomical profile, which objectively confirmed the effectiveness of the chosen therapy.

The pictorial documentation played a crucial role in monitoring the course of the pathology and assessing the effectiveness of treatment, and represented an important tool in the comprehensive management of the patient with chorioretinitis.

In the long-term monitoring of the patient, which at present is ongoing for one year, stable central visual acuity is maintained at a level of 20/25. The subjectively perceived visual deficit, originally described as a dark band in the central part of the visual field, was reduced during the course of therapy, and practically disappeared after the treatment was discontinued. The patient has no complaints or sign of recurrence of the pathology, and has functional preserved vision.

DISCUSSION

Chorioretinitis is a significant ophthalmological complication upon neuroinfection caused by herpes simplex virus type 1 (HSV-1), in which its precise occurrence depends on the specific study and the observed population. Experimental studies using a mouse model have demonstrated a high incidence of HSV retinopathy, which developed in as many as 91% of inoculated eyes and 88% of non-inoculated eyes six days after inoculation. In the case of congenital HSV infection, chorioretinitis is con-

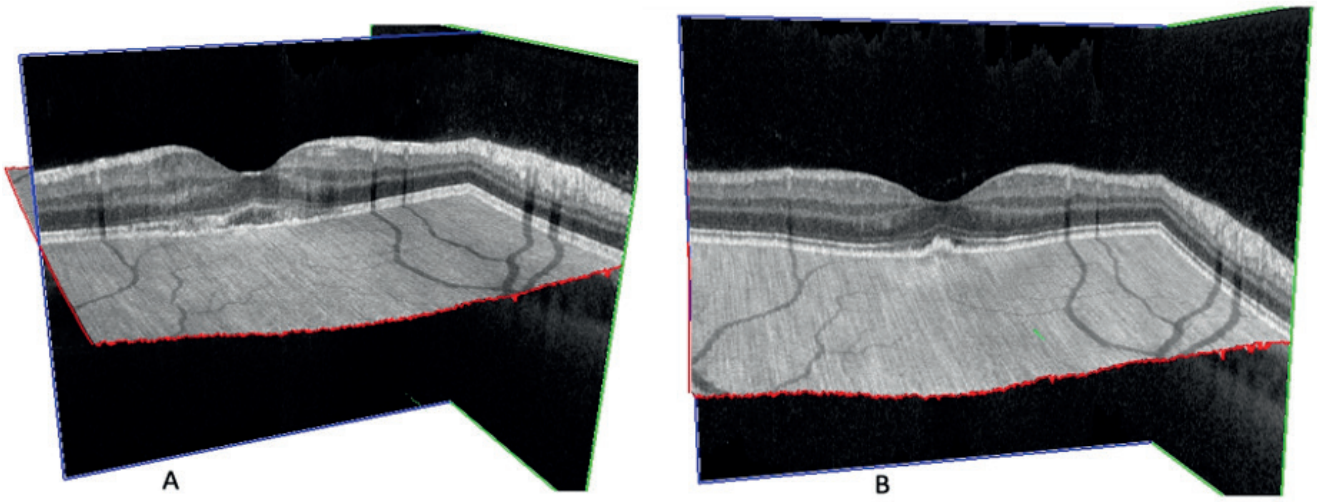

Figure 6. Three-Dimensional Optical Coherence Tomography (3D OCT) imaging of the retina demonstrating disease progression and treatment response

sidered one of the most common ocular manifestations [11,12]. The clinical manifestations of ocular complications upon a background of HSV-1 infection have been widely described in the professional literature. Retinitis caused by HSV-1 frequently presents as necrotizing retinitis with vascular inflammation and optic neuropathy, with minimal vitritis [13].

This case report illustrates a rare but significant correlation between reactivation of latent herpes simplex virus type 1 (HSV-1) and previous infection with parvovirus B19, which probably played a role in weakening the patient's immune system. The interaction between parvovirus B19 and reactivation of latent HSV-1 has not been examined in detail in the professional literature. The mechanisms leading to the reactivation of HSV-1 include impairment of immunological balance, which may be induced by various viral infections, including parvovirus B19 [14]. This virus is known for its capacity to influence the cellular and humoral immune response, which may create the conditions for reactivation of latent viruses [15–18]. A case study such as this one indicates the potential synergic effect between the viruses and opens up questions relating to their mutual interactions, highlighting the need for a deeper understanding of immunological processes that influence the reactivation of latent infections. The lack of epidemiological data concerning the incidence of chorioretinitis upon a background of these coinfections accentuates the need for prospective trials. In its result, this case indicates the need for a multidisciplinary approach and a better understanding of virus interactions, which could influence the diagnosis and management of similar cases.

Within the framework of differential diagnostics it was necessary also to consider the possibility of a pathology with a similar clinical picture and similar findings from the imaging examinations, including acute macular neuroretinopathy (AMN), unilateral acute idiopathic maculopathy (UAIM) and Vogt–Koyanagi–Harada (VKH) syndrome.

AMN is typically manifested in acute appearance of paracentral scotomas in young patients, in which OCT demonstrates localized lesions in the outer retinal layers and FAG as a rule does not show significant dye bleeding [19,20]. UAIM has a unilateral course with a characteristic image on FAG and OCT, often after viral prodrome [21,22]. VKH syndrome is a multisystemic autoimmune disorder with specific bilateral ocular and systemic manifestations, including a typical finding on FAG and OCT [23,24].

In our case several clinical trials and imaging characteristics did not correspond to these diagnoses, which enabled us to exclude them. A detailed comparison of the key symptoms, findings on FAG and OCT and the reasons for exclusion of these pathologies is presented in Table 1.

In the presented case, after diagnosis intravenous treatment with acyclovir 750 mg every 8 hours over the course of 14 days was indicated, followed by oral treatment of 400 mg 2× per day over the course of 6 weeks. The procedure and length of treatment were consulted with the Department of Infectology and Geographical Medicine at the Faculty of Medicine of Comenius University, and adapted to the patient's clinical condition.

Cases have been described in the professional literature in which acute retinal necrosis has developed following herpetic infection of the CNS, even after a substantial length of time [25,26]. This supports the case for prolonging antiviral treatment in certain cases. On the other hand, long-term prophylaxis with acyclovir may lead to reduced specific cellular immunity to HSV, with a decrease in the proliferation of HSV-specific lymphocytes and a slight (~10 %) decrease in the levels of IgG antibodies [27,28]. Some studies also point to the risk of development of resistant strains upon prolonged treatment [29].

Table 1. Comparison of selected entities included in the differential diagnosis with the presented case

Disease	Typical clinical manifestations	FA findings	OCT findings	Reason for exclusion in the presented case
Acute macular neuroretinopathy (AMN) [Aziz et al., 2015; Fang et al., 2023]	Sudden onset of paracentral scotomas, often in young women, associated with infections or systemic diseases	Hypofluorescent lesions in both early and late phases of FA	Hyperreflective lesions in the deep retinal layers (outer plexiform and nuclear layers) involving photoreceptors	The location and character of the changes on OCT and FA were not typical for AMN; the course was not spontaneously regressive, and the patient had confirmed neuroinfection
Unilateral acute idiopathic maculopathy (UAIM) [Nicolo et al., 2016; Qiu et al., 1998]	Acute vision loss, often after viral infection, unilateral involvement	Hyperfluorescence in the late phases with possible pooling of dye	Thickening of the neurosensory retina, subretinal fluid, damage to the outer photoreceptor layers	In our case, there was significant cystoid macular edema together with subretinal fluid, which in its extent and characteristics does not correspond to the typical presentation of UAIM
Vogt–Koyanagi–Harada syndrome (VKH) [Lavezzo et al., 2016; Tayal et al., 2024]	Bilateral granulomatous panuveitis, systemic manifestations (meningismus, tinnitus, vitiligo, poliosis), often in patients with pigmented skin	Multiple hyperfluorescent spots with late-phase dye leakage, pooling of dye in the subretinal space	Subretinal fluid, septations, diffuse involvement of the RPE	In our case, there was no bilateral involvement, no systemic manifestations, and no typical OCT pattern of VKH

FA – fluorescein angiography

The decision to prolong treatment should therefore be based on an individual assessment of the risk of reactivation, the clinical course, presence of risk factors and tolerance to treatment, in which it is necessary to ensure long-term ophthalmological monitoring, which applies in the presented case.

CONCLUSION

This clinical case highlights the importance of a multidisciplinary approach in the diagnosis and treatment of complex pathologies. It places emphasis on the interconnection of ophthalmological, neurological and laboratory observations in managing rare and complex pathologies such as chorioretinitis within the context of HSV-1 neuroinfection. Timely identification of the symptoms, integrated application of modern diagnostic technologies and interdisciplinary cooperation brought about effective diagnosis and commencement of the correct therapy, thereby leading to the desired positive result.

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