CLINICAL OBSERVATIONS

Use of olokizumab in giant cell arteritis with rapidly progressive ischemic optic neuropathy (clinical case)

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We present a clinical case of giant cell arteritis (GCA) with ophthalmic manifestations (anterior ischemic optic neuropathy) successfully treated with the direct interleukin-6 inhibitor olokizumab in combination with glucocorticoids. This case demonstrates the efficacy of early initiation of biologic drugs for rapid control of inflammatory activity and prevention of irreversible vision loss in GCA.

Keywords: *giant cell arteritis; anterior ischemic optic neuropathy; olokizumab; interleukin-6 inhibitor.*
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Giant cell arteritis (GCA) is a systemic vasculitis of large and medium-sized vessels that primarily affects individuals over 50 years of age. The peak incidence occurs at 70–80 years of age, with prevalence in women 2–3 times higher than in men [1]. The etiology of the disease is not fully understood, but a key role in the pathogenesis is played by dendritic cells of the vascular wall, which activate T-lymphocytes with subsequent production of pro-inflammatory cytokines such as interleukin 6 (IL6). IL6 is the key mediator of the systemic inflammatory response in GCA, which justifies the therapeutic strategy of its blockade [2, 3].

The clinical manifestations of GCA are diverse and range from the classic cranial variant with headache, temporal artery pathology and jaw "claudication" to atypical forms with predominantly constitutional symptoms [4, 5]. The most serious complication of the disease is ischemic optic neuropathy, which leads to irreversible blindness. Vision loss in GCA can develop rapidly and is the debut manifestation of the disease in 15–20% of cases [6]. The mechanism of blindness development is associated with granulomatous inflammation and occlusion of the posterior short ciliary arteries that provide blood supply to the optic nerve, which clinically manifests as anterior ischemic optic neuropathy [6, 7].

Glucocorticoids (GCs) which should be prescribed immediately when GCA is suspected remain the "gold standard" of therapy [8]. However, long-term use of GCs is associated with the development of adverse events, especially in elderly patients with comorbid pathology [9]. In addition, some patients are refractory to therapy or have relapses of the disease when the GCs dose is reduced [9, 10]. In this regard, the possibilities of steroid-sparing therapy are being actively studied. To date, the IL-6 receptor inhibitor tocilizumab (TCZ), which is included in international recommendations, has the largest evidence base [8, 11].

Olokizumab (OKZ) is a humanized monoclonal antibody of the immunoglobulin G4/kappa isotype that binds to IL-6 and blocks its pro-inflammatory effects. In the Russian Federation, the product is registered for the treatment of rheumatoid arthritis (RA) [12]. However, accumulating data from clinical series demonstrate its potential efficacy in other IL6-dependent diseases, including GCA and polymyalgia rheumatica [2].

We present the clinical case of a female patient with severe GCA complicated by rapidly progressive vision loss, in whom the use of OKZ not only reversed systemic inflammation but also preserved vision in the only seeing eye. The patient gave consent for her medical data to be published.

Clinical case

Female patient T.*, 83 years old, developed acute symptoms in October 2025 with a rise in temperature to febrile levels (maximum – up to 38.5 ℃). A few days later she manifested with an intense headache, pain in the eyeballs, severe pain when chewing, and swelling in the temporal regions. During visit to ophthalmologist, visual acuity was 1.0 in both eyes, but magnetic resonance imaging of the brain revealed edema in the subarachnoidal space of the right optic nerve. The neurologist prescribed mannitol without clinical improvement, which indirectly confirmed the absence of isolated neurological pathology and the presence of systemic inflammation. On 23/10/2025, the patient noted a sharp decrease in vision in the right eye, that can be described as a "curtain-like visual disturbance," followed by complete loss of vision.*

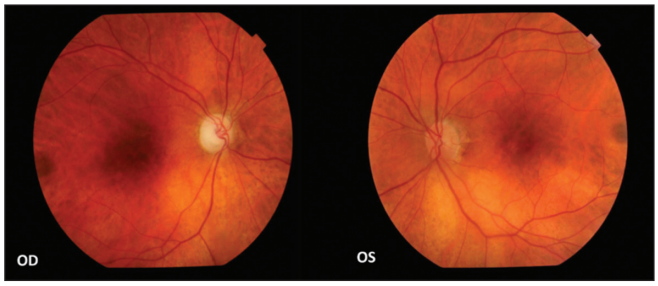

Fig. 1. *Ophthalmoscopy findings in patient T.*

In November 2025, she was hospitalised in the ophthalmology department. Upon admission, she complained of loss of vision in her right eye, progressive loss of vision in her left eye, headache, pain in the temporal region, sharp pain when chewing, and increased body temperature.

Ophthalmoscopy revealed signs of ischemic neuropathy. In the right eye (OD), the optic nerve disc (OND) is pale, chalky-white, pasty, the borders are blurred, the arteries are narrowed. In the left eye (OS), the OND is pale pink, the superotemporal border is blurred, edema of the peripapillary retina, ischemic foci, and splinter hemorrhages are detected (Fig. 1). Optical coherence tomography (OCT) confirmed ischemic edema and decreased number of retinal ganglion cells on the right and thickening of the nerve fibre layer on the left (Fig. 2). Duplex scanning revealed a critical decrease in blood flow in the central arteries, in the posterior short ciliary arteries (especially on the right), which is consistent with literature data that posterior ciliary arteries are affected in GCA [13, 14]. A significant increase in ESR (up to 63 mm/h) and CRP levels (up to 90.2 mg/L) was noted. Taking into account the ischemic nerve damage, pulse therapy with methylprednisolone (500 mg No. 3) was started with positive dynamics such as alleviation of headache and a decrease in temperature, but without vision improvement. The patient continued to report decreased vision in her left eye. A paroxysm of atrial fibrillation (AF) developed during GC therapy; it was stopped by amiodarone.

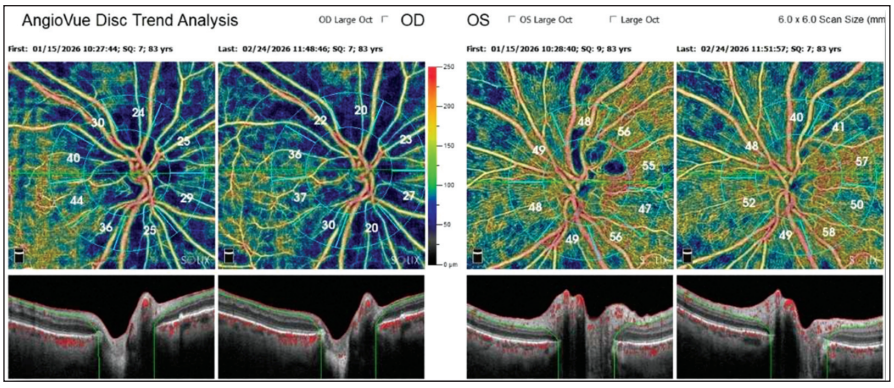

Fig. 2. *OCT findings in patient T.*

The patient was examined by a rheumatologist. The general condition is satisfactory. Body temperature is 37.2 ℃ The skin is pale and dry. During examination, temporal arteries on both sides were bulging and thickened, as well as tender on palpation.

She was transferred to the rheumatology department, where prednisolone 30 mg/day orally was prescribed.

During therapy, a repeated paroxysm of AF occurred (11/11/2025), and episodes of a marked increase in blood pressure (BP) were noted.

Computed tomography angiography (CTA) of the neck vessels was performed; it revealed circular thickening of the walls of the descending aorta (Fig. 3, a, b), the right subclavian artery (Fig. 3, c, d) and stenosis of the left vertebral artery secondary to thickening of its walls (Fig. 3Q, d, e).

Based on the ACR/EULAR (American College of Rheumatology/European Alliance of Associations for Rheumatology) 2022 criteria [15], a diagnosis of GCA was established: involvement of the descending aorta, right subclavian artery, left vertebral artery, temporal arteries, central retinal arteries, posterior short ciliary arteries; intermittent claudication of the masticatory muscles, high activity.

Considering the progression of optic nerve ischemia on the left with the risk of total blindness, high disease activity and the impossibility of increasing the dose of GCs due to cardiac pathology (AF paroxysms, unstable blood pressure), the expert panel decided to prescribe a biological disease modifying antirheumatic drug (bDMARD). The decision of the expert panel determined the need for emergent off-label initiation of the direct IL-6 inhibitor OKZ because of the technical availability of the drug product, as well as based on its comparable efficacy with TCZ in the treatment of RA [16] and the emerging positive results of the use of OKZ in GCA [2].

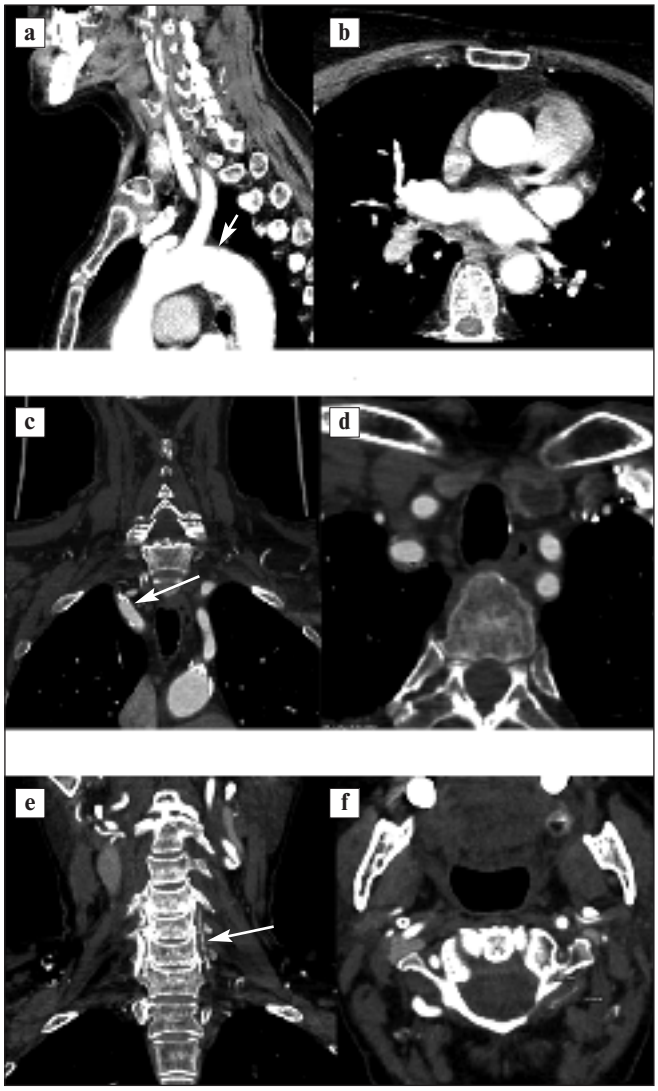

Fig. 3. *CT angiography (CTA) of the neck vessels in patient T.: a, b – circumferential thickening of the descending aortic wall (arrow); c, d – circumferential thickening of the right subclavian artery wall (arrow); e, f – stenosis of the left vertebral artery against the background of circumferential wall thickening (arrow)*

An examination was carried out according to the "safety profile": determination of anti-HIV antibodies; HBsAg; anti-HCV antibodies;

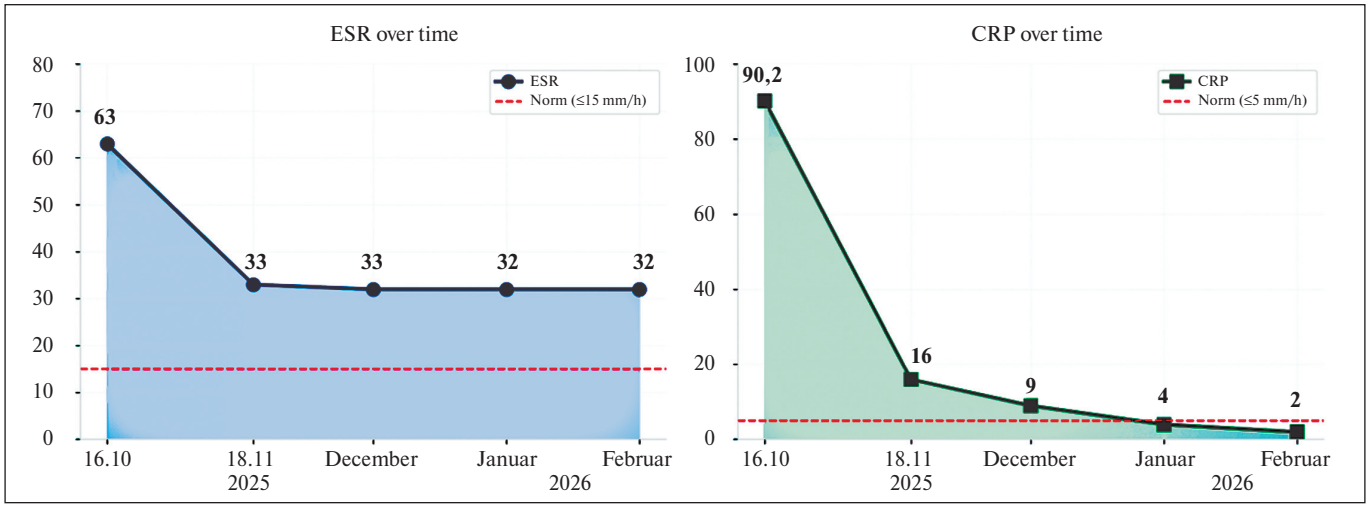

Fig. 4. *Changes in ESR (mm/h) and CRP level (mg/L) during therapy*

Diaskintest; chest X-ray. There were no contraindications for the use of bDMARD.

On 13/11/2025, therapy with OKZ was initiated at a dose of 64 mg. After 5 days of treatment, pain in the temporal region and pain when chewing completely subsided, changes in the temporal artery regressed, and vision in the left eye stabilized. Over the course of 1.5 months, the dose of prednisolone was reduced until it was completely discontinued due to cardiac pathology.

OKZ administration was continued every 28 days: in December 2025, January and February 2026. A stable trend towards a decrease in inflammation markers was noted: by February 2026, the CRP level decreased to 2 mg/L (Fig. 4).

During a follow-up examination by an ophthalmologist in February 2026, only punctate ischemia and a single hemorrhage were noted on the fundus of OS; according to OCT data, the thickness of the OS retina was normal. Left eye vision has stabilised.

Final diagnosis. *Major disease: GCA – involvement of the descending aorta, right subclavian artery, left vertebral artery, temporal arteries, central retinal arteries, posterior short ciliary arteries; intermittent claudication of the masticatory muscles, high activity. Complications: anterior ischemic optic neuropathy of the left eye. Atrophy of the optic nerve of the right eye (consequences of anterior ischemic neuropathy).*

Concomitant diseases: ischemic heart disease, arrhythmic variant – paroxysmal form of AF, paroxysms dated 03 and 11/11/2025, restoration of sinus rhythm dated 03 and 11/11/2025. CHA2DS2-VASc 3b, HAS-BLED 3b. EHRA IV. Chronic heart failure stage I, functional class I. Hypertension stage III, partially controlled arterial hypertension. Left ventricular hypertrophy. Hypertensive nephrosclerosis. Chronic kidney disease C3a (glomerular filtration rate according to CKD-EPI – 48.5 mL/min/1.73 m²). Risk of cardiovascular complications 4 (very high). Target BP: 130–139/70–79 mm Hg.

Discussion. The presented clinical case demonstrates a typical picture of GCA with severe ophthalmic manifestations. The patient had a classic triad of symptoms: headache in the temporal region, tenderness when chewing and decreased vision, which, in combination with a marked increase in acute phase indicators, made it possible to make a diagnosis without performing a temporal artery biopsy [17].

Ophthalmic complications of GCA remain one of the most serious issues in clinical practice. Anterior ischemic optic neuropathy develops as a result of occlusion of the posterior ciliary arteries that supply the anterior part of the optic nerve [6, 7].

Timely diagnosis and immediate initiation of high-dose GCs therapy are key factors determining the prognosis for vision in GCA [8, 9]. According to literature, in the absence of treatment, the risk of developing bilateral blindness reaches 60% [6, 18]. In our case, therapy was started from the moment of the patient's admission, which made it possible to prevent further progression of ischemia and maintain partial visual acuity.

It is interesting to note that the patient had lesions of the aorta and its large branches during CTA. Circular thickening of the walls of the descending aorta, subclavian and vertebral arteries indicates the systemic nature of vasculitis. Damage to large vessels in GCA is associated with a more severe course of the disease and requires long-term follow-up due to the risk of developing aortic aneurysms [19].

The initiation of OKZ was justified by the need for rapid control of inflammatory activity and reduction of the GCs dose. The results of CREDO clinical program demonstrated the high efficacy of OKZ in RA, including in patients with failure of therapy with other bDMARDs. The drug product showed a good safety profile, comparable to that of other IL-6 inhibitors [20]. The results of real-world evidence studies confirmed the high effectiveness of OKZ in reducing clinical and laboratory signs of disease activity, as well as the possibility of rapid discontinuation of GCs in more than 50% of patients or a reduction in their doses [21, 22].

The laboratory parameters over time demonstrate a rapid decrease in CRP and ESR levels during therapy. 5 days after the first administration of OKZ, a decrease in the CRP level by 82% (from 90.2 to 16 mg/L) was already observed, which indicates the high effectiveness of the drug product in suppressing the inflammatory process. By February 2026, the CRP level was completely normalised (2 mg/L), which made it possible to discontinue GCs. Good tolerability of OKZ was noted. Given the known risks of IL6 inhibition (risk of diverticulitis/intestinal perforation, neutropenia, increased liver transaminase levels) [20, 22] and the patient's advanced age, she was informed of symptoms requiring attention (abdominal pain, fever, weakness, etc.). During the observation period, no adverse events or complications were registered.

The choice of bDMARD therapy duration for GCA remains controversial. In the GiACTA study, TCZ was administered for 52 weeks [23]. Due to the high risk of recurrence of GCA and the presence of large vessel lesions in our patient, long-term therapy with OKZ with regular monitoring of laboratory parameters and instrumental examination data is planned.

Conclusion. Thus, the presented clinical case demonstrates the efficacy of therapy with a direct IL6 inhibitor OKZ in GCA with ophthalmological complications. Early administration of bDMARD allows for rapid control of inflammation and a reduction in the dose of GCs, which is especially important for elderly patients with a high risk of developing adverse reactions to GCs.

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Conflict of Interest Statement

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