Atypical Optic Neuritis: Case Presentation

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Summary. *Purpose.* To present a case report with "unclear" loss of visual acuity in the left eye.

Methods. A case report.

Case presentation. A 42-year-old woman presented to the ophthalmology department with a 30-day history of double vision and 3 days absolute visual acuity loss and a relative afferent pupillary defect in her left eye. Visual acuity in her left eye was 0 (no perception of the light) and her right eye visual acuity, Farnswoth-Munsell 100 hue color vision test, and visual field were normal. Slit-lamp examination of the anterior segment was otherwise normal but sub-acute intermediate uveitis was noticed. Optic nerve discs of both eyes were normal. The patient had not multiple sclerosis diagnosis in anamnesis and no changes were found in magnetic resonance imaging one month before. She had cervical cancer 14 years ago treated by radiotherapy. Computerised tomography scan procedure was done in the hospital but no changes were found. Ultrasound imaging was done as well. Only slight thickening of the medial and inferior muscles were noticed, optic nerve without changes. No changes were found in the abdomen by ultrasound. Chest x-ray showed residual changes after tuberculosis. Magnetic resonance imaging of the head and the orbits was repeated in the hospital and revealed no pathological changes.

Intravenous methylprednisolone therapy for four days was administered. After four days treatment the left eye visual acuity was 0.03.

Conclusion. In presenting this clinical case, we would like to present an atypical optic neuritis clinical case with double vision as first symptom one month prior and acute loss of visual acuity as the primary symptom of atypical optic neuritis.

Keywords: atypical optic neuritis, visual functions.

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INTRODUCTION

The term optic neuritis (ON) is used to define an acute, demyelinating, or idiopathic optic neuropathy. The average onset of the disease is at 30 years of age, and it afflicts women more often than men (a ratio of approximately 2:1) [1]. Typical ON is characterized by a loss of vision that develops over days and is associated with dyschromatopsia, visual field loss, and pain that is often exacerbated by eye movements, and it may occur in up to two-thirds of individuals suffering from multiple sclerosis at some point in the course of their disease [2, 3]. Usually there are no retinal exudates or severe disc swelling and vision is better than "no light perception" [2]. The classic presentation of ON is unilateral loss of vision in the majority of adults. The vision loss is quite variable and ranges from mild to no light perception [4]. ON includes infectious and inflammatory aetiologies [1]. The most important factor is probably the loss of signal transmission in some axons due to a conduction block or ganglion cell death [5]. It is likely due to some inflammatory process which leads to delayed type IV hypersensitivity reaction induced by released cytokines and other inflammatory mediators from activated peripheral T-cells which can cross the blood brain barrier and cause destruction of myelin, neural cell death and axonal degeneration [6]. Mostly visual acuity in patients with optic neuritis at the first attack ranges from 0.1 to 0.3.

So in this article we would like to present a case report with loss of visual acuity without light perception in the left eye due to atypical optic neuritis.

CASE REPORT

A 42-year-old woman presented to the Ophthalmology Department, Lithuanian University of Health Sciences, Kaunas Clinic, with a 30-day history of double vision and 3 days absolute visual acuity loss without light perception and relative afferent pupillary defect in her left eye. The patient was bitten by a mite two months prior. Antibodies Ig M and IgG of Lyme disease were used twice: first time results showed that Ig G was doubtful; and the second time IgG and IgM were negative. One month after the mite

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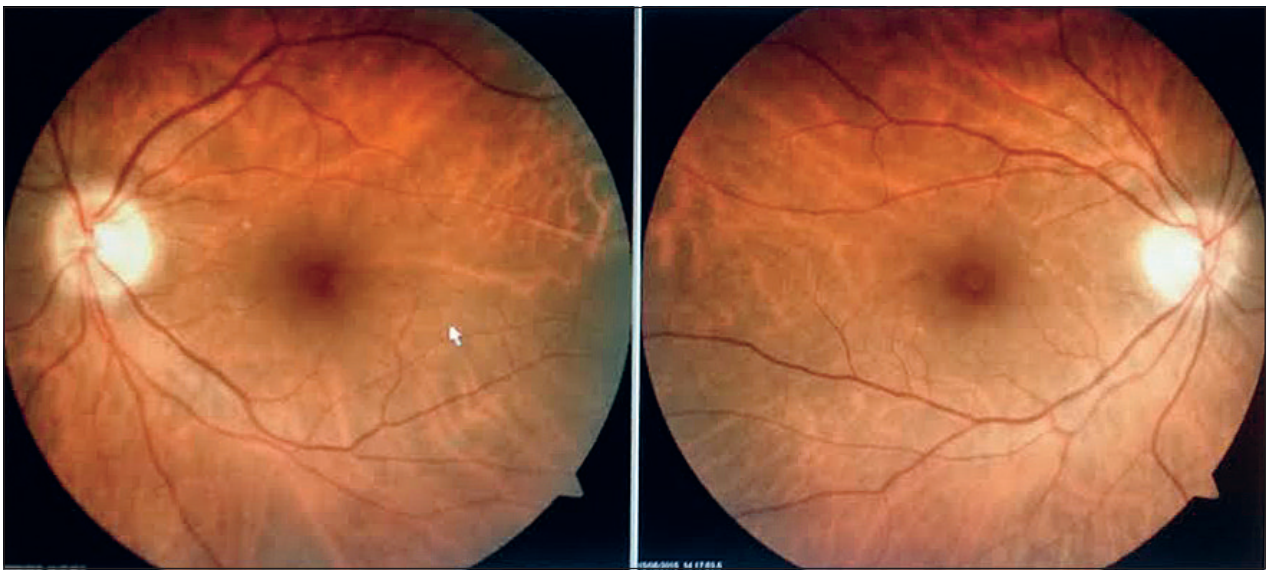


Fig. 1. **Fundus photography of both eyes**

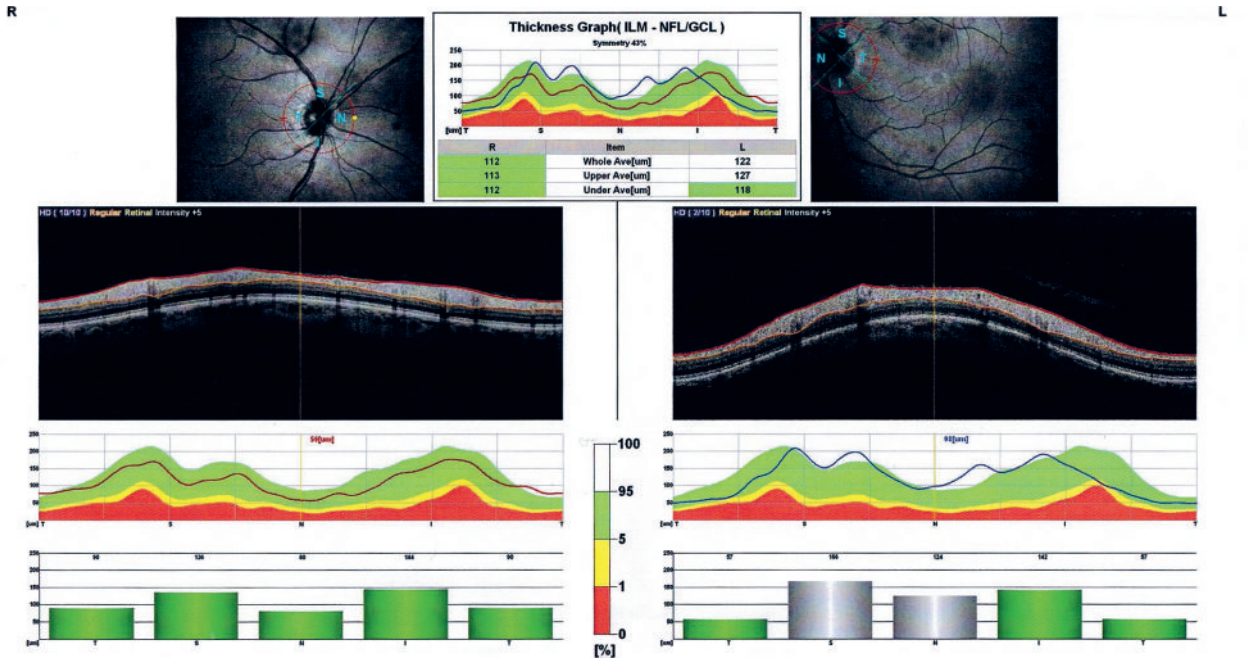


Fig. 2. **Optical coherent tomography**

bite the patient noticed red spots on the body that disappeared but occurred in another area of the body. This symptom lasted about one week and later disappeared. Visual acuity in her left eye was 0 (no light perception), and in her right eye visual acuity, Farnswoth-Munsell 100 hue colour vision test, and visual field were normal. Ocular pressure in the right eye was 17.3 mm/Hg, and in the left eye – 15.9 mm/Hg. Slit-lamp examination of the anterior segment was normal but the sub-acute intermediate uveitis was noticed. In periphery parts of vitreous, "snowball" form white spots were noticed. Optic nerve discs of both eyes were normal (Fig. 1). Ultrasound imaging was done as well. Only slight thickening of the medial and inferior muscles were noticed. Optic nerve discs were without pathological changes. Optical coherent tomography of the right eye was normal but examination of the left eye revealed retinal nerve fiber layer swelling of top and nasal quadrants (Fig. 2). Perimetry of the right eye showed visible 121 points from 135 points, of the left eye visible 0 point from 135 points (Fig. 3). Visual evoked potentials examination revealed: both eyes p100 waves were deformed, the left is misjudge, and the right eye – normal (Fig. 4).

Retrobulbar blood flow examination showed – slightly increased left eye artery resistance (the right eye – OA – 0.71; CRA – 0.47; +SPCA – 0.53; the left eye – OA – 0.77; CRA – 0.48; +SPCA – 0.47).

The patient had not multiple sclerosis diagnosis in anamnesis and no changes were found in magnetic resonance imaging one month before. She had cervical cancer

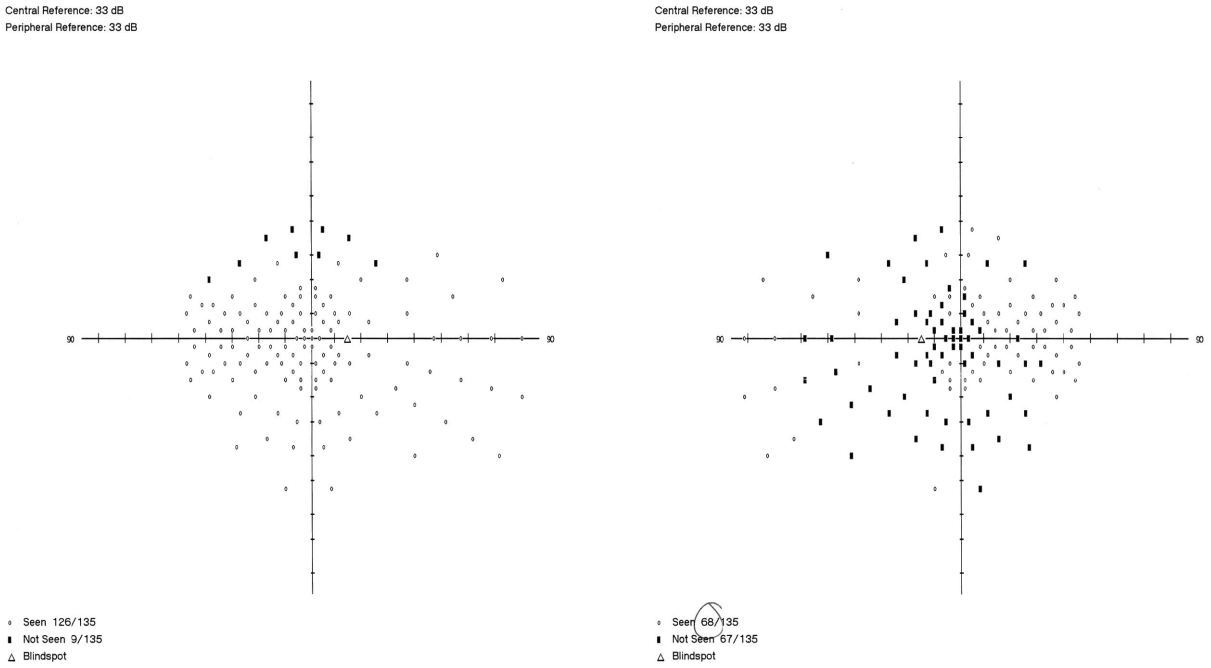
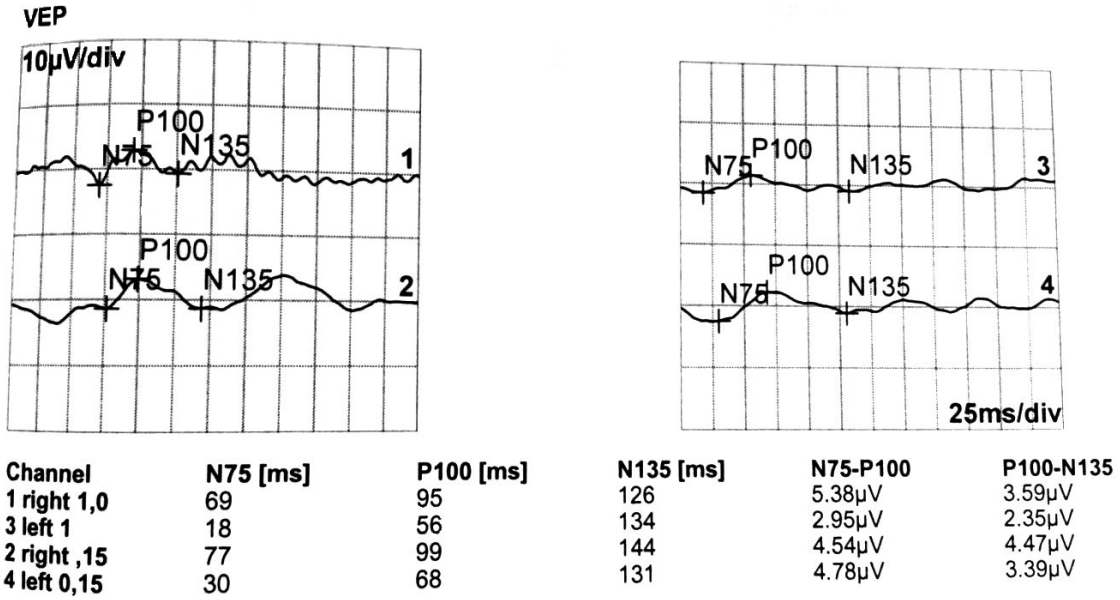


Fig. 3. **Perimetry of both eyes**



Channel	N75 [ms]	P100 [ms]	N135 [ms]	N75-P100	P100-N135
1 right 1,0	69	95	126	5.38µV	3.59µV
3 left 1	18	56	134	2.95µV	2.35µV
2 right ,15	77	99	144	4.54µV	4.47µV
4 left 0,15	30	68	131	4.78µV	3.39µV

Fig. 4. Visual evoked potentials

14 years ago and was treated by radiotherapy. Computerised tomography scan procedure of the head was done in the hospital but no changes were found as well. No changes were found in the ultrasound of the abdomen. Chest x-ray showed residual changes after tuberculosis. Magnetic resonance imaging of the head and the orbit was repeated once again, and revealed no pathological changes.

Intravenous methylprednisolone 1000 mg. therapy for three days was administered. After three days treatment the left eye visual acuity recovered to 0.03.

As a slight improvement of visual acuity was achieved after intravenous methylprednisolone therapy we think about atypical optic neuritis diagnosis which first symptom was double vision one month prior and acute absolute blindness without light perception as the first symptom of optic neuritis.

Treatment with intravenous Solumedrol (Methylprendnizolon) 1000 mg. for 3 days was prescribed, later followed by tab. Prednisoloni 60 mg., decreasing to 5 mg., which 5 days. Eye drops Dexa-Chlora 5 times per day in the left eye were administered. Tab. Ranitidini 150 mg. 1 time per day were prescribed for elevated stomach acidity.

Visual acuity outcome. After 3 intravenous infuses visual acuity in the left eye was – 0.02, after one month – 0.05, and after two months – 0.06. Perimetry after one month was done once again, and 68 points from 135 points were seen (Fig. 5).

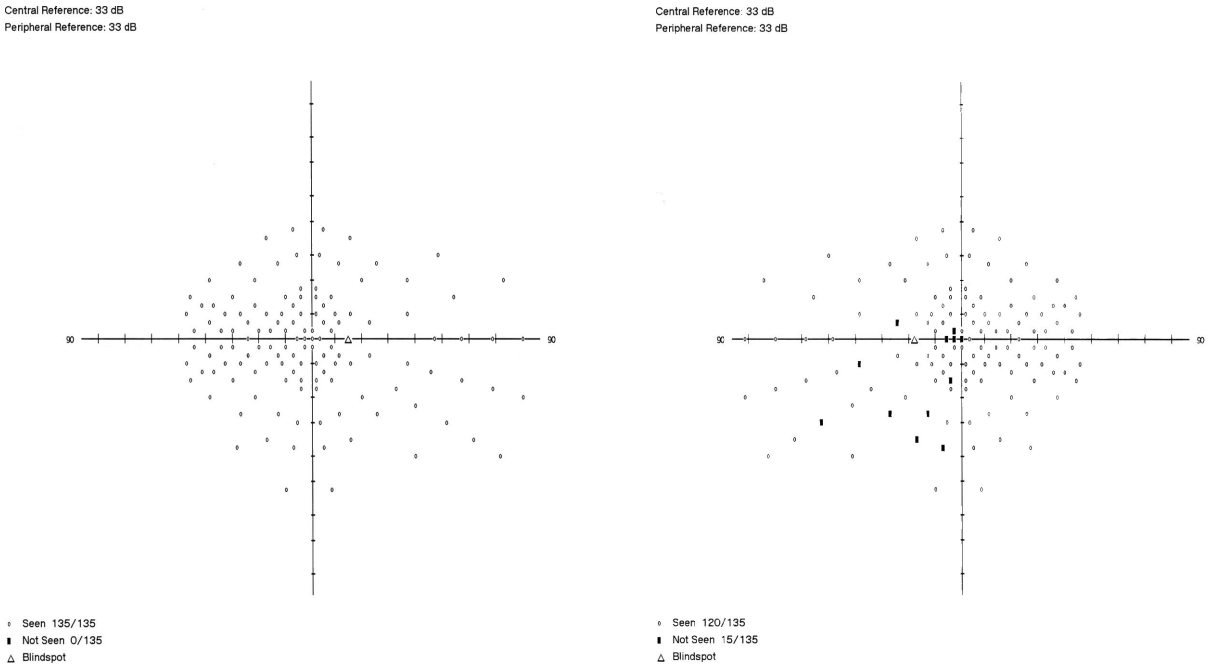


Fig. 5. Perimetry

DISCUSSION

The classic presentation of ON is unilateral loss of vision in the majority of adults. ON mostly occurs in persons under 50 years old and ratio of females to males is 3.5:1 [7]. The vision loss is quite variable and ranges from mild to no light perception but typically it ranges from 0.1 to 0.3. Typically there is a short period of progression over hours to 10 days followed by improvement: complete or almost complete recovery of visual acuity and visual field are common [8]. Progressive visual worsening for more than 2 weeks or lack of recovery after 8 weeks, should suggest an alternative diagnosis to demyelinating (MS type) ON [9]. Periocular pain and retro-orbital pain occurs in >90% of cases, usually exacerbated by eye movement, may precede or coincide with the visual symptoms, and usually resolves over days. Most patients show reduced contrast sensitivity and dyschromatopsia, which are often out of proportion to the visual acuity deficit. When colour loss is present, most persons show mixed red–green and blue–yellow colour defects [10].

The cause of typical ON is unknown. As it is often associated with MS, it is thought that the two conditions share a common etiology. There are probably complex interactions between genetic predisposition and environmental triggers, that have yet to be identified but may include infectious agents and vitamin D deficiency [11]. Atypical ON may be caused by infections, or be related to other inflammatory and autoimmune diseases. Causes of atypical neuritis is presented in Table 1 [12]. Some diseases can mimic optic neuritis, and are presented in Table 2.

In most cases, visual recovery will occur spontaneously but i.v. steroids can speed the rate of recovery. There is an association of ON with MS and baseline MRI imaging provides important prognostic information for MS. Typi-

Table 1. **Causes of atypical neuritis**

Etiology	Cause of ON
Inflammatory	• Neuromyelitis optica • Sarcoidosis • SLE • Behçet's • Chronic relapsing inflammatory optic neuropathy • Autoimmune ON • Acute disseminated encephalomyelitis
Infectious	• Lyme • Syphilis • Viral (EBV, CMV, HIV, others) • Tuberculosis • Toxoplasmosis • Toxocariasis • Bartonella (cat scratch) • Histoplasmosis
Postinfectious	• Commonly children, bilateral

Table 2. **Diseases mimics of optic neuritis**

Anatomical region	Condition
Retina	• Central serous retinopathy (CSR) • Big blind spot syndrome/acute zonal occult outer retinopathy
Sclera	• Posterior scleritis
Optic nerve	• Ischemic optic neuropathy (may be related to giant cell arteritis – in which case may be painful – or diabetic papillitis) • Optic nerve compression • Leber's hereditary optic neuropathy • Toxic optic neuropathy • Nutritional optic neuropathy • Drug-induced
Orbit	• Orbital cellulitis
Other	• Functional

cal ON can usually be diagnosed clinically, and does not require routine laboratory testing, however atypical ON may require further tests to rule out secondary causes of ON.

Normal vision is recovered in 50 to 78% of the patients within six months. However, in some cases ON produces long-lasting objective and/or subjective visual dysfunction [13]. As in our case, visual acuity recovered to 0.06.

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ATIPINIO REGOS NERVO NEURITO KLINIKINIO ATVEJO PRISTATYMAS

Santrauka

42 metų moteris kreipėsi į oftalmologą dėl 30 dienų trunkančio dvejinimosi ir dėl 3 dienas trunkančio regos netekimo bei aferentinės vyzdžio reakcijos kairėje akyje. Regos aštrumas kaire akimi buvo 0 (šviesos juslės nebuvo), matymas dešine akimi, Farnswoth-Munsell atspalvių tyrimas ir akipločio tyrimas buvo nepakitę. Apžiūrint plyšine lempa priekinį segmentą, pakitimų nenustatyta, bet, apžiūrint stiklakūnį, nustatytas poūmis vidurinis uveitas kairėje akyje. Regos nervų diskai abiejų akių – be pakitimų. Taip pat anamnezėje nebuvo diagnozuota išsėtinė sklerozė, nenustatyta pakitimų galvos smegenyse magnetinio rezonanso tomografijos tyrime, kuris buvo atliktas prieš 1 mėnesį. Prieš 14 metų pacientei buvo diagnozuotas gimdos kaklelio vėžys, pravestas spindulinis gydymas. Ligoninėje atlikta galvos smegenų kompiuterinė tomografija, bet pakitimų nebuvo nustatyta. Atlikus ultragarsinį tyrimą, nustatytas kairės akies medialinio ir apatinio raumenų nedidelis sustorėjimas; regos nervas – be pakitimų. Taip pat atlikta ir viršutinio pilvo aukšto echoskopija – pakitimų nenustatyta. Atlikus krūtinės ląstos rentgenogramą, nustatyti liekamieji pakitimai po persirgtos tuberkuliozės. Galvos smegenų ir orbitų magnetinis rezonansas dar kartą buvo pakartotas ligoninėje, bet pakitimų galvos smegenyse nenustatyta.

Atlikus minėtus tyrimus, taikytas gydymas pulsterapija metilprednizolonu (1 gramas tris dienas), vėliau tęstas gydymas prednizolonu per os. Po keturių dienų gydymo matymas kaire akimi atsistatė iki 0,03 po buvusio visiško aklumo.

Pristatydami šį klinikinį atvejį, norime pateikti atipinio regos nervo neurito klinikinį atvejį, kurio pirmas simptomas – dvejinimasis, prasidėjęs prieš 1 mėnesį, ir ūmus regėjimo aštrumo netekimas, kuris pasireiškė kaip pirminis atipinio neurito simptomas.

Raktažodžiai: atipinis regos nervo neuritas, regos funkcijos.

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