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Severe acute bilateral alcohol-induced toxic optic neuropathy – case report

Ciężka ostra obustronna alkoholowa toksyczna neuropatia nerwu wzrokowego – opis przypadku

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Streszczenie: Wstęp: chociaż neuropatie toksyczne nie są zbyt często spotykane w praktyce klinicznej, stanowią jednak wyzwanie zarówno w sensie diagnostyki, jak i leczenia. Celem pracy jest prezentacja przypadku zatrucia alkoholem etylowym, które spowodowało obustronną neuropatię toksyczną z utratą widzenia.
Opis przypadku: przedstawiamy przypadek obustronnej utraty widzenia spowodowanej alkoholową neuropatią toksyczną nerwu wzrokowego.
Słowa kluczowe: toksyczna neuropatia nerwu wzrokowego, etanol, alkohol.
Summary: Toxic optic neuropathies are not frequently encountered in routine practice, however, they present a challenge both in terms of diagnosis and treatment. The aim of this paper is to present an unusual case of ethyl alcohol poisoning causing bilateral toxic optic neuropathy with loss of vision.
Key words: toxic alcohol-induced optic neuropathy, ethanol, alcohol.

Introduction

Toxic optic neuropathies are not frequently encountered in routine practice, however, they present a challenge both in terms of diagnosis and treatment. The clinical findings of toxic optic neuropathies share some similarities with nutritional deficiency optic neuropathies (1).

Both methanol and ethanol are well known optic nerve toxins. Alcohol optic neuropathy has different clinical picture than other toxic optic neuropathies. Ethanol is usually associated with tobacco-alcohol-induced toxic optic neuropathy, whereas methanol causes acute, severe, and permanent visual loss and in severe cases it may even lead to death (1).

The aim of this paper is to present an unusual case of ethyl alcohol poisoning and toxic optic neuropathy with bilateral loss of vision which resulted from the intoxication.

Case report

A 32 years old Caucasian male was admitted to the department of ophthalmology because of sudden, bilateral, painless loss of vision, without any other accompanying ophthalmic symptoms. The patient confirmed that he was an alcohol addict and that he abused alcohol frequently and admitted to having abused alcohol (ethanol only) for the last two weeks continuously, on the daily basis. According to the patient, after drinking alcohol for two weeks, his vision deteriorated within one day. At that day he had nausea and stomach ache. He did not want to see the doctor at this time, as he thought his condition was temporary and would resolve spontaneously. When his vision deteriorated, he withdrew alcohol, however, it was as long as 5 days after his vision had deteriorated when he went to seek medical advice.

On initial examination the patient presented with bilateral severe deterioration of visual acuity. Best corrected distance visual acuity (BCDVA) amounted to hand movements in both

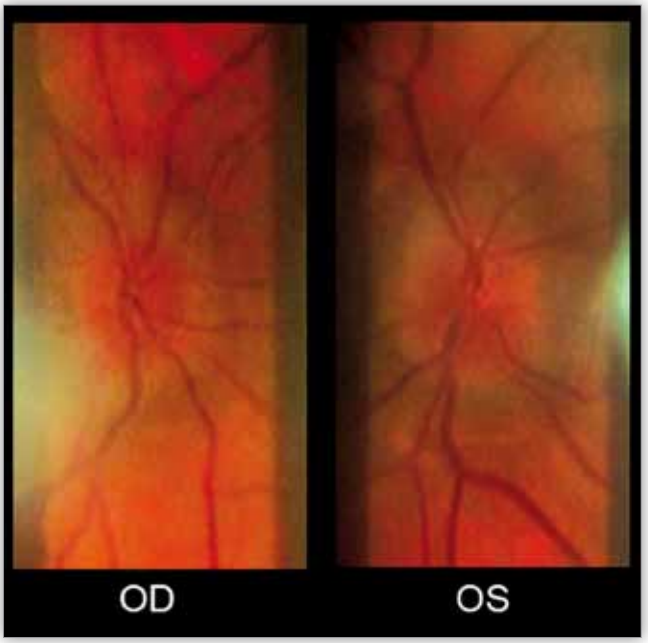

Fig. 1. Optic discs on admission (slit-lamp photos).
Ryc. 1. Tarcza nerwu wzrokowego w badaniu wykonanym podczas przyjmowania pacjenta (obraz z lampy szczelinowej).

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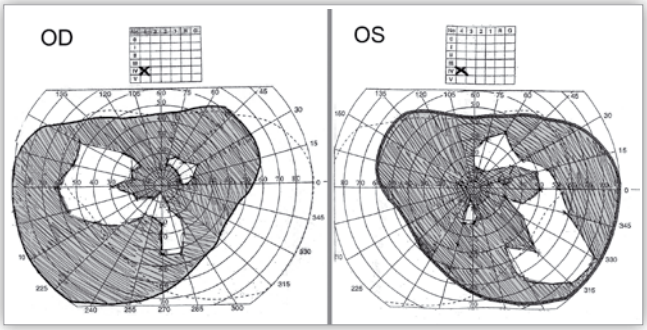

Fig. 2. Goldmann kinetic perimetry.
Ryc. 2. Perymetria kinetyczna Goldmanna.

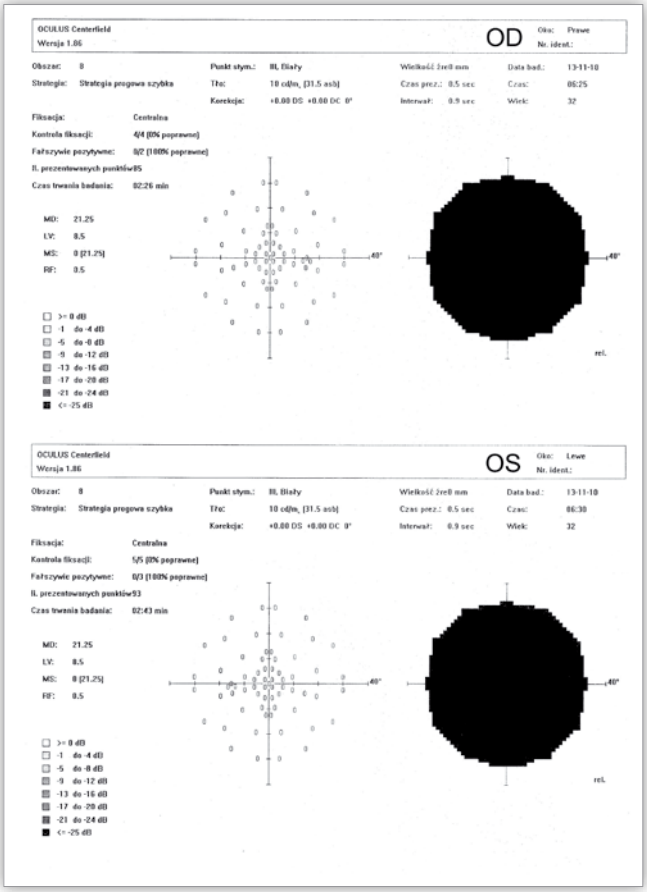

Fig. 3. Computerized static perimetry.
Ryc. 3. Komputerowa perymetria statyczna.

eyes. Intraocular pressure (IOP) measured by aplanation Goldmann tonometry was 7 mmHg bilaterally.

The biomicroscopic examination revealed a normal anterior segment, pupils were equal, round, slowly reacting to light. The fundus examination revealed edematous and engorged optic nerve discs, normal retinal vessels and pale and edematous central retina with intraretinal haemorrhages (which were more prominent in the left eye) (Fig. 1). Patient denied having dyschromatopsia.

His previous ophthalmological and general medical history was unremarkable, as was his family medical history. The patient denied using any medicines, as well as any history of trauma.

On admission, the patient was consulted with a neurologist, who did not find any other signs (no nystagmus, no ataxia, no headache, no nausea, no dizziness, normal Romberg sign, etc.) and recommended planned head imaging examinations.

Goldmann visual perimetry was performed, which revealed significant visual field defects with temporal islands of vision (Fig. 2).

The treatment included general steroids (Encorton 50 mg/day) and intravenous electrolytes. The next day static perimetry was performed, showing severe loss of vision and incorrect fixation (Fig. 3).

After 2 days, fundus fluorescein angiography (FA) (and colour fundus photograph) were performed (Fig. 4, 5). FA showed

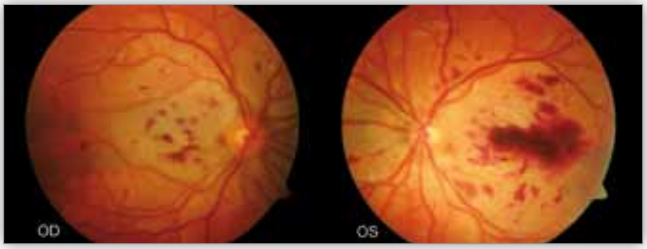

Fig. 4. Color fundus photograph from the fundus camera.
Ryc. 4. Fotografia kolorowa dna oka – obraz z fundus kamery.

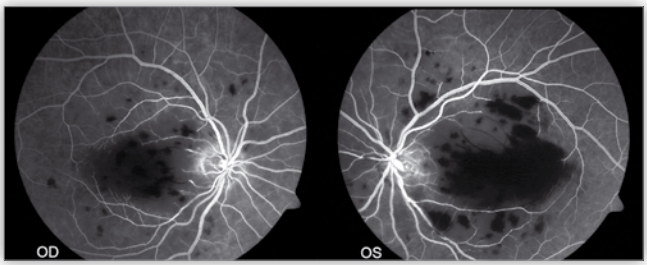

Fig. 5. Fluorescein angiography.
Ryc. 5. Angiografia fluoresceinowa.

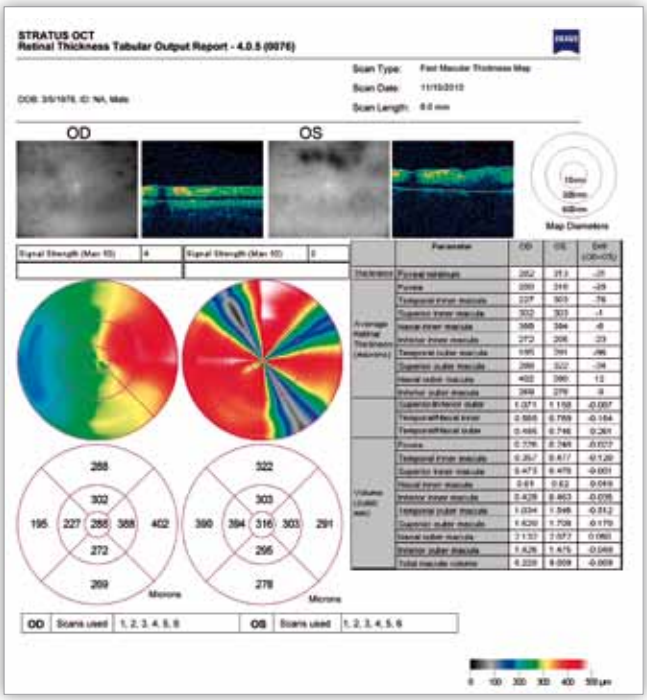

Fig. 6. OCT of the macula (fast macular thickness map).
Ryc. 6. OCT plamki (tryb "fast macular thickness map").

areas of hypofluorescence in the edematous macular area, as well as intrareretinal haemorrhages.

Optical coherent tomography (OCT) of the macula revealed edema and lack of normal retinal profile (Fig. 6). Retinal nerve fiber layer (RNFL) measurement showed greatly increased thickness in all quadrants in both eyes, indicating RNFL edema (Fig. 7).

Visual evoked potentials (VEP) showed axonal lesions, decreased amplitude of potentials and increased P100 latency and were strongly indicative of severe damage to both visual pathways (Tab. I).

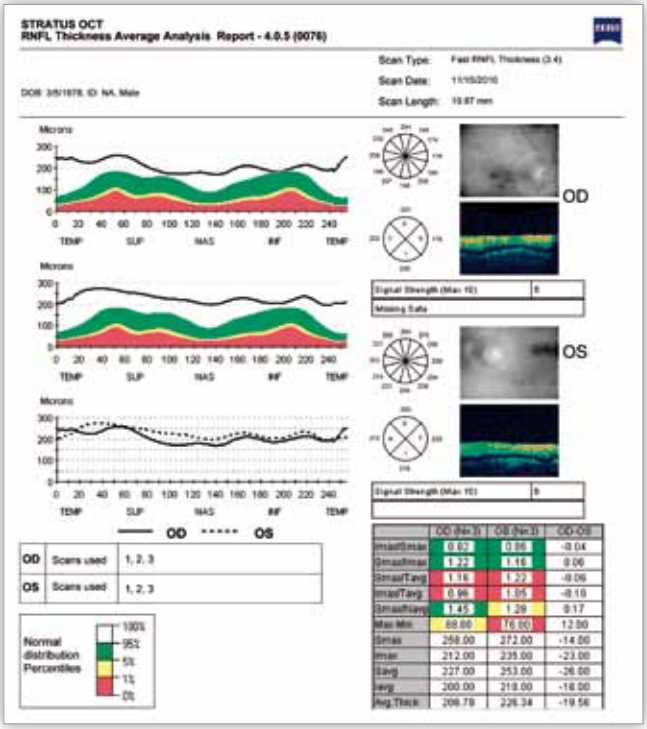

Fig. 7. RNFL thickness measurement done by OCT (Fast RNFL thickness).
Ryc. 7. Pomiar grubości włókien nerwowych RNFL w OCT (tryb „Fast RNFL thickness").

Protocol/ Run	N75 ms	P100 ms	N145 ms	P100 μV	Size
VEP					
L – VEP	no response				
R – VEP	no response				
1.1 Oz – Fz	100.75	**125.25**	153.00	**0.18**	16
1.2 O1 – Fz	97.00	**127.50**	155.00	**2.0**	
1.3 O2 – Fz	104.00	**129.00**	155.75	**2.0**	
2.1 Oz – Fz	98.50	**125.00**	154.00	**1.5**	16
2.2 O1 – Fz	94.50	**126.25**	142.00	**2.6**	
2.3 O2 – Fz	106.50	**127.25**	154.00	**0.07**	

Tab. I. Result of VEP examination.
Tab. I. Comparison of suspension materials.

After a week the patient decided to stop the treatment, he did not agree to have any other examinations done and discharged himself from the hospital, despite being warned of possible negative consequences.

Discussion

In cases of optic neuropathies with visual-field defects the differential diagnosis includes: other toxic amblyopias, Leber's hereditary optic neuropathy, retrobulbar optic neuritis (demyelinative), and perichiasmatic tumors (1).

There are a few substances which are generally thought to be causes of toxic optic neuropathies, they include: arsenicals, Carbon disulfide, Disulfiram, Ethylene glycol, Lead, Ethanol, Methanol, Tobacco, some drugs (e.g. Amiodarone, Dapsone, Ethambutol, Isoniazid, Quinine, Streptomycin) and other substances. Usually laboratory tests of blood and urine are useful for confirming the exposure to a particular toxin and they help to establish the correct diagnosis. In any intoxication it is crucial to stop the exposure and recognize the agent, both in order to treat the patient appropriately and to prevent others from being exposed. Early treatment includes correction of the acidosis, or even hemodialysis. Our patient did not have acidosis, his blood alcohol level was not tested, as he had withdrawn alcohol five days earlier (1).

Nowadays, ethanol-induced optic neuropathy is not frequently reported. Clinical picture of chronic alcoholic neuropathy may include: peripheral neuropathy (e.g. limb weakness, numbness, diminished tendon reflexes) and optic neuropathy. Visual loss is painless and bilateral, it can be of various severity. Loss of central vision may be chronic or subacute, it is usually accompanied by decreased visual acuity and dyschromatopsia. Some individuals lose vision rapidly and a usual late consequence is optic atrophy which develops after one to two months and leads to total blindness. Visual field test characteristically shows central and centrocecal scotomas. In severe cases, pupillary dilatation can be found, it usually signifies a poor prognosis, as it occurs in almost total or total blindness. The eye fundus may appear normal, although there may be abnormalities, especially atrophy of the optic disc. In acute intoxication the optic discs may appear swollen and hyperemic, the edema may extend to the surrounding retina (1,2).

In contrast, methanol-induced optic neuropathy occurs sporadically, it is usually present in an alcoholic who has consumed the toxin. If ethyl alcohol is consumed at the same time, it reduces the toxicity by competing for alcohol dehydrogenase (1). Clinical picture of toxic methanol-induced optic neuropathy is well-known and may include: headache, dyspnea, nausea and vomiting, abdominal pain, dyschromatopsia, bilateral central visual field defects and bilateral visual impairment in a normal looking eye (even visual loss). Frequently these symptoms commence 1 to 2 days after the intoxication (1-6). Animal studies have shown that methanol intoxication causes mainly retrobulbar optic neuropathy with disc edema and these findings were confirmed in human autopsies (6-10).

Our patient had a form of optic neuropathy resembling both ethanol and methanol-induced neuropathy. He denied drinking methyl alcohol, however, he admitted to having had a long two weeks period of continuous drinking ethyl alcohol. Our patient

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had a significant visual loss and marked visual field defects, however, he denied dyschromatopsia.

Our findings are in accordance with some previous reports describing RNFL thickness measurements above normal limits due to RNFL edema in patients with alcohol-induced toxic optic neuropathy. In contrast, chronic use of tobacco and alcohol is associated with the nerve fiber layer loss, which in some cases may be partially reversible or irreversible (11,12).

Hantson et al. (13) found, that during the early stage of methanol poisoning about a half of patients had early signs of reversible retinal dysfunction, in another half persistent electrophysiologic signs of optic neuropathy were found. The occurrence of optic neuropathy and early electrophysiologic data (visual evoked potentials) were correlated.

Moura et al. (14) described RNFL measurements done with OCT scanning in 3 patients with progressive visual loss resulting from chronic alcoholism. In two patients there was RNFL loss in the temporal sector of the optic disc and in one patient RNFL thickness was above normal limits, possibly due to RNFL edema similar to the one that may occur in the acute phase of toxic optic neuropathies. In our patient OCT revealed bilateral thickening above normal limits, suggesting a massive edema of optic nerve fibers.

Previously, abnormalities in the electroretinogram (ERG) were described in a monkey model of ethanol-induced toxic optic neuropathy. It is thought that these changes reflect the diminished retinal function associated with the visual abnormalities observed in alcohol-induced optic neuropathy (15). Our patient did not have ERG, however, VEP examination revealed bilateral severe damage of the optic tract.

Nutritional optic neuropathies are uncommon and are usually associated with gradual visual loss and optic atrophy. The clinical findings in the toxic optic neuropathies are similar to the symptoms of nutritional deficiency neuropathies. Both these diseases share some symptoms, moreover, in some patients it is not possible to tell whether neuropathy was caused by toxic or nutritional cause (1). Nutritional optic neuropathies usually have the form of non-specific, bilateral, painless, chronic and slowly progressive retrobulbar optic neuropathy (16).

Optic disc edema is one of the differences between these two entities – it is commonly found in toxic neuropathies, however, it is rarely present in nutritional deficiency amblyopia. Another difference is the possible reversibility of optic neuropathy resulting from Vitamin B_{12} and folate deficiencies, in contrast to methanol-induced toxic optic neuropathy which is usually irreversible (1).

Metabolic toxic optic neuropathies are distinguished from degenerative neuropathies by nutritional deficiencies (e.g. folic acid, vitamin B_{12} deficiency as well as protein-energy malnutrition). It is thought that nutritional deficits are associated with the development of toxic alcohol-induced neuropathy, so their separation into two components is artificial (2,16).

Recovery from alcohol-induced optic neuropathy may be possible after refraining from alcohol completely and using vitamin supplements, although, Paparrigopoulos et al. (2) described a case of irreversible visual loss due to alcohol abuse. As our patient denied having ever used methanol, we assume that his symptoms were associated with heavy drinking of ethanol, however, we cannot be certain of what kind of alcohol he abused. We do not have long-term observations of our patient, as he discharged himself and discontinued treatment, so we do not have any information as to possible visual recovery.

Shimozono et al. (17) described a case of nutritional optic atrophy with permanent vision loss, central scotoma, and an acquired red-green color vision defect. They concluded that alcohol-induced nutritional optic neuropathy should not be viewed as an isolated ocular entity, but rather as a potentially treatable neurologic problem.

In many cases, it is also necessary to test blood samples for the mitochondrial DNA mutations that have been found in patients with Leber's hereditary optic neuropathy (1,18). Leveziel et al. (19) emphasized that Leber's optic neuropathy must be suspected in a recent decrease in visual acuity, even if it appears in a context of alcohol and tobacco intoxication.

Bilateral vision loss may also result from compression or infiltration of the chiasm (they can cause bilateral central or centrocecal scotomas). In patients with bilateral visual loss, head imaging is also indicated, however, our patient did not agree to have it done, as well as refused to having any other tests performed.

There are papers reporting that the administration of high doses of intravenous steroids followed by oral steroids leads to an improvement of vision in patients with methanol-induced optic neuropathy, if the interval between the consumption of methanol and starting treatment is short (20). In our patient steroid treatment diminished optic disc edema, however, it did not influence visual acuity.

Even if optic neuropathy resulting from ethanol poisoning is treated in a proper and timely manner, the patient may still experience permanent neurologic sequelae. Intense ethyl alcohol abuse may lead to alcohol-induced toxic optic neuropathy, and as a result may cause bilateral loss of vision.

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