(19)

Nutritional optic neuropathy

Neuropatia wzrokowa powstała wskutek niedoborów pokarmowych

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Summary: Nutritional optic neuropathy (aka deficiency optic neuropathy) is a dysfunction of the optic nerve resulting from improper dietary content of certain nutrients essential for normal functioning of the nerve fibers. Most commonly, it results from folic acid and vitamin B complex deficiency associated with malnutrition or poor dietary habits, incorrectly applied vegetarian diet, or chronic alcohol abuse. Obese patients after bariatric surgery constitute another risk group of optic neuropathy. Nutritional optic neuropathy is characterized by painless, gradually progressing, bilateral and symmetrical decrease in visual acuity, which can be accompanied by the color vision dysfunction. Progression of the neuropathy is associated with optic nerve atrophy, manifesting as complete disc pallor. Treatment of nutritional neuropathy includes dietary supplementation, aimed at compensating for the deficient nutrients. The treatment is mostly based on folic acid, vitamin B complex, and protein replacement, as well as eliminating risk factors of neuropathy. Early treatment commencement, prior to irreversible optic nerve atrophy, is a prerequisite of effective treatment.
We would like to highlight this problem by presenting the case of a young woman in whom chronic use"water-based" diet resulted in anemia and bilateral nutritional optic neuropathy.

Key words: optic neuropathy, nutritional deficiency, toxic neuropathy.

Streszczenie: Neuropatia wzrokowa powstała wskutek niedoborów żywieniowych jest zaburzeniem funkcji nerwu wzrokowego spowodowanym niewłaściwą ilością określonych składników pokarmowych w diecie, niezbędnych do prawidłowego funkcjonowania włókien nerwowych. Najczęściej jest ona wywołana niedoborem kwasu foliowego i witamin z grupy B w wyniku niedożywienia lub złych nawyków żywieniowych, nieprawidłowo stosowanej diety wegetariańskiej bądź przewlekłego alkoholizmu. Inną grupę ryzyka stanowią chorzy otyli po leczeniu bariatrycznym. Neuropatia ta charakteryzuje się wolno postępującą, bezbolesną, obustronną i symetryczną utratą widzenia, towarzyszy temu zaburzenie widzenia barwnego. W miarę rozwoju neuropatii dochodzi do zaniku nerwów wzrokowych, przejawia się to całkowitym zblednięciem tarcz nerwów wzrokowych. W leczeniu neuropatii z niedoborów pokarmowych stosuje się odpowiednią dietę suplementacyjną, wyrównującą brakujące składniki pożywienia. Opiera się ona głównie na podaży kwasu foliowego, witamin z grupy B i białka, a także na eliminacji czynników ryzyka, które doprowadziły do rozwoju neuropatii. Warunkiem skuteczności leczenia jest szybkie podjęcie terapii, zanim dojdzie do nieodwracalnego zaniku nerwów wzrokowych.
Chcieliśmy podkreślić wagę tego problemu poprzez prezentację przypadku młodej kobiety, u której w wyniku długotrwałego stosowania diety „wodnej" doszło do rozwoju niedokrwistości oraz neuropatii niedoborowej obu nerwów wzrokowych.

Słowa kluczowe: neuropatia wzrokowa, niedobory pokarmowe, neuropatia toksyczna.

Causes and risk factors

Nutritional optic neuropathy is a functional disorder of the optic nerve resulting from the dietary deficiency of certain nutrients required for normal functioning of nerve fibers. This type of neuropathy can develop due to the insufficient dietary intake of vitamin B_1 (thiamin), vitamin B_{12} (cobalamin), folic acid, vitamin B_6 (pyridoxine), vitamin B_2 (riboflavin), and vitamin B_3 (niacin) (1). Extremely rarely, the neuropathy can develop due to copper deficiency (2). The exact mechanism of neuropathy development associated with the vitamin deficiency has not been completely understood, but it is postulated that the deficiency impairs biochemical processes at a cellular level. The inadequate intake of vitamin B_{12} and/or folic acid promotes synthesis and accumulation of toxic formate, which block the process of oxidative phosphorylation in mitochondria. This leads to the decreased synthesis of adenosine triphosphate (ATP), excessive accumulation of reactive oxygen species, and disorders of axonal transport in the optic nerve fibers.

Apart from obvious nutritional reasons, there are also certain risk factors that increase the likelihood of optic neuropathy development in susceptible individuals. These include alcohol abuse and cigarette smoking, both of which can cause nicotine-alcohol neuropathy with clinical manifestations nearly identical as the nutritional neuropathy. Consequently, many authors classify these conditions as a single entity, referred to as "toxic neuropathy". Chronic alcohol abuse is usually associated with eating disorders resulting from the replacement of other energetic sources with highly caloric alcohol. Heavy alcohol users are usually malnourished, sometimes even cachectic, and their diet does not contain enough proteins and vitamins, eventually leading to nutritional neuropathy. In turn, cyanide contained in tobacco smoke exerts a toxic effect on the optic nerve fibers, which is enhanced further by vitamin B complex deficiency. Therefore, one may ask whether it is nicotine-alcohol neuropathy or rather toxic / nutritional neuropathy in such cases (3).

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Epidemiology

The nutrient deficiency should persist for at least several months to manifest as nutritional optic neuropathy. The risk of neuropathy is not affected by age, gender, or race; however, it can be increased by such stimulants as alcohol or nicotine. Depending on the cause, the optic nerve disorders can manifest as isolated episodes or epidemic outbreaks. Some geographical regions, such as Cuba, Tanzania, Gambia, Somalia, Nigeria, and Papua New Guinea, are characterized by high incidence of this neuropathy (4–9). Nutritional deficiencies resulting from poor economic situation, war conflicts and weather anomalies (drought, storm, flood) were postulated as the main causes contributing to such high number of cases of this ophthalmic disorder.

Apart from epidemic outbreaks of deficiency optic neuropathy, sporadic cases usually associated with the deficiency of vitamin B_1, B_{12}, and/or folic acid, or rarer other B complex vitamins have been reported. Typically, these disorders result from poorly balanced diet, e.g. during slimming treatment, or using various restriction diets, including vegetarian diet and its numerous varieties. These diets are based on the exclusion of meat, fish, and eggs from the diet, all being the only nutritional sources of vitamin B_{12}. Some more radical diets allow solely for the consumption of unprocessed fruits and vegetables. When followed for longer periods, such diets wreck tremendous havoc in the body as it is impossible to compose meals containing all necessary nutrients of allowed products (10, 11).

Obese patients after bariatric surgery constitute another risk group of optic neuropathy. Gastric reduction surgeries usually lead to the deficiency of vitamin B_1, B_6, B_{12}, folic acid, vitamin D and E, and copper (12, 13). Overall, as many as 16% of patients subjected to various types of bariatric surgery exprience neurological and/or ophthalmic complications. Nutritional deficiencies result from impaired intestinal absorption and highly restrictive protein-rich and fat-deficient diet recommended postoperatively.

Moreover, vitamin B_{12} deficiency can be a consequence of malignant anemia associated with the presence of autoantibodies against the parietal cells of gastric mucosa. This is reflected by the vitamin B_{12} malabsorption from the ileum and its severe deficiency.

Clinical manifestation

Nutritional optic neuropathy is characterized by painless, gradually progressing, bilateral and symmetrical decrease in visual acuity, which can be accompanied by color vision dysfunction. Frequently, dyschromatopsia is the initial sign of this condition. The color vision dysfunction is typically not proportional to the degree of vision loss and can manifest as perceiving colors as darker or "dirty", or as the complete loss of color vision. The patient describe the initial signs of vision loss as a centrally located spot. Subsequently, visual acuity decreases gradually, although it is rarely impaired to the level of counting fingers in front of the face. These changes are accompanied by subtle impairment of pupillary light reflexes, which can be misinterpreted as normal due to their symmetric nature. Wide, non-reactive pupils are observed at the terminal stages of the condition after the complete atrophy of the optic nerves has occurred.

Fundus abnormalities depend on the stage of the disease. At initial stages of the neuropathy, the optic discs can appear normal, sometimes slightly swollen and/or hyperemic with small hemorrhages. Neuropathy progression is associated with optic nerve atrophy, manifesting as temporal disc pallor (Fig. 1), or, at the terminal stage, as complete disc pallor. Visual field testing usually shows neuropathy-specific central or centrocecal scotoma with perimetry sparing (Fig. 2). Peripheral visual field narrowing or altitudinal scotomas are extremely rare. Analysis of visually evoked potentials shows abnormal nerve fibre conductivity, manifested as a considerable decrease in wave amplitude. The wave latency is usually normal or only slightly prolonged. Changes in perimetry and visual evoked potentials are usually symmetrical.

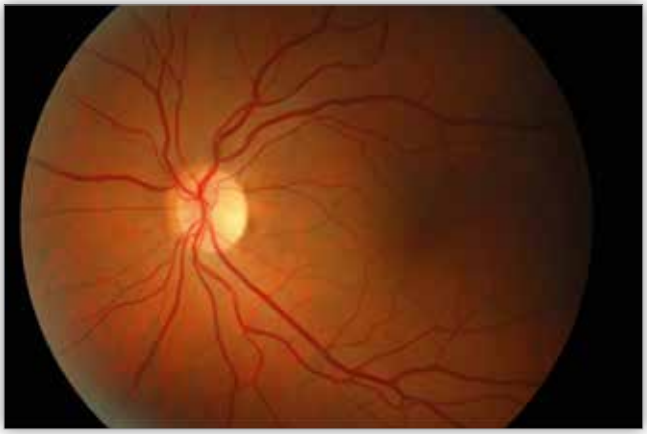

Fig. 1. Partial optic disc atrophy in nutritional neuropathy (temporal pallor of the optic disc).
Ryc. 1. Częściowy zanik tarczy nerwu wzrokowego w przebiegu neuropatii niedoborowej (skroniowe zblednięcie tarczy nerwu wzrokowego).

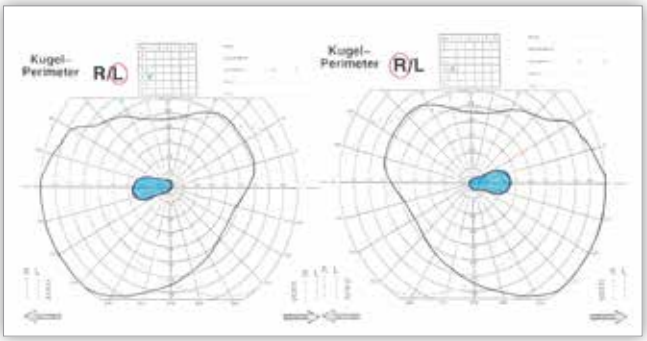

Fig. 2. Kinetic perimetry – bilateral centrocecal scotoma.
Ryc. 2. Kinetyczne pole widzenia – obuoczne mroczki centrocekalne.

Diagnosis

History taking plays a vital role in the evaluation of nutritional optic neuropathy. It should focus on everyday diet, dietary restrictions, ingested stimulants and drugs, and exposure to certain harmful factors. Many substances and prescription drugs are considered toxic to the optic nerve; the complete list along with use and sources is presented in tables I–IV. Knowledge of chemicals and medications which can be toxic to the optic nerve is vital for the differential diagnosis of optic neuropathy, especially since the symptoms of classical toxic neuropathy and nutritional neuropathy are virtually identical.

Furthermore, certain stages of many other neuropathies, including late post-traumatic neuropathy, retrobulbar neuro-

Amiodarone/ Amiodaron	Anti-arrhythmic agent/ czynnik antyarytmiczny
Amoproxan/ Amoproxan	Anti-arrhythmic agent/ czynnik antyarytmiczny
Cafergot/ Cafergot	Drug used in migraine attacks/ środek leczniczy używany w leczeniu ataków migreny
Quinine/ Chinina	Alkaloid used in treatment of malaria/ alkaloid używany w leczeniu malarii
Chloramphenicol/ Chloramfenikol	Antibiotic / antybiotyk
Chlorpromazine/ Chlorpromazyna	Antipsychotic agent/ czynnik antypsychotyczny
Chlorpropamide/ Chlorpropamid	Agent stimulating insulin synthesis; used in the treatment of diabetes/ czynnik stymulujący syntezę insuliny, używany w leczeniu cukrzycy
Dapsone/ Dapson	Agent used in the therapy of dermatologic conditions/ lek używany w leczeniu zaburzeń dermatologicznych
Disulfiram/ Disulfiram	Agent used in chronic alcoholism; trade name: Esperal/ lek stosowany w leczeniu przewlekłego alkoholizmu, nazwa handlowa: Esperal
Deferoxamine/ Deferoksamina	Used in the therapy of acute iron intoxication, trade name: Desferal/ używany w leczeniu ostrego zatrucia żelazem; nazwa handlowa: Desferal
Ethambutol/ Etambutol	Agent used in the treatment of tuberculosis/ lek stosowany w leczeniu gruźlicy
Ethchlorvynol/ Ethchlorv ynol	Sedative and hypnotic agent; trade name: Placidyl/ lek uspakajający i nasenny, nazwa handlowa: Placidyl
Infliximab/ Infliximab	Drug uses in treatment of Crohn's disease; trade name: Remicade/ lek używany w leczeniu choroby Crohna; nazwa handlowa: Remicade
Interferon α/ Interferon α	Protein with antiviral and anticancer agent/ białko o działaniu przeciwwirusowym i przeciwnowotworowym
Isoniazid/ Izoniazyd	Drug used in the treatment of tuberculosis/ lek używany w leczeniu gruźlicy
Penicillamine/ Penicylamina	Drug used in treatment of Wilson's disease and cystinuria, and in thallium, mercury, lead, and cadmium intoxication/ lek używany w leczeniu choroby Wilsona i cystynurii, oraz zatruć talem, rtęcią, ołowiem i kadmem
Sildenafil/ Sildenafil	Used in the treatment of erectile disorders; trade name: Viagra/ lek używany w leczeniu problemów z erekcją; nazwa handlowa: Viagra
Streptomycin/ Streptomycyna	Aminoglycoside antibiotic; used in the treatment of tuberculosis/ antybiotyk aminoglikozydowy używany w leczeniu gruźlicy
Sulphonamids/ Sulfonamidy	Antibacterial agents/ lek antybakteryjny
Tamoxifen/ Tamoksyfen	Anti-estrogenic agent used in the therapy of breast cancer/ środek antyestrogenowy stosowany w leczeniu raka piersi

Tab. I. Drugs with potential toxic effect on the optic nerve.
Tab. I. Leki o potencjalnie toksycznym wpływie na nerw wzrokowy.

Chlorambucil/ Chlorambucyl	Used in the therapy of chronic lymphatic leukemia/ stosowany w terapii przewlekłej białaczki limfatycznej
Cisplatin/ Cisplatyna	Used in anticancer chemotherapy/ używana w chemioterapii nowotworów
Ciclosporin/ Cyklosporyna	Used after transplantation and in treatment of psoriasis and atopic dermatitis/ używana po przeszczepach oraz w leczeniu łuszczycy i atopowego zapalenia skóry
5-fluorouracil/ 5-fluorouracyl	Cytostatic agent used in the anticancer therapy/ środek cytostatyczny używany w leczeniu nowotworów
Tacrolimus/ Takrolimus	Used after transplantation and in the treatment of atopic dermatitis/ stosowany po przeszczepach oraz w leczeniu atopowego zapalenia skóry
Vincristine/ Winkrystyna	Used in the therapy of leukemia, Hodgkin lymphoma, multiple myeloma, neuroblastoma, and small-cell lung cancer/ używana w leczeniu białaczki, choroby Hodgkina, szpiczaka mnogiego, neuroblastomy oraz raka drobnokomórkowego płuc

Tab. II. Cytostatics and immunosuppressive drugs with potential toxic effect on the optic nerve.
Tab. II. Cytostatyki i leki immunosupresyjne o potencjalnie toksycznym wpływie na nerw wzrokowy.

pathy, posterior ischemic neuropathy, congenital optic nerve atrophy, or Leber's neuropathy, can have a similar manifestation. Therefore, careful inquiry about the patient's potential exposure to any harmful substances can facilitate making the right diagnosis. Additional toxicological tests, detecting both toxins and their metabolites in tissues and body fluids,

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Carbon tetrachloride/ Czterochlorek węgla	Solvent of pigments and glues; previously used in tetra fire extinguishers/ rozpuszczalnik barwników i kleju, początkowo używany w gaśnicach
Dinitrobenzene/ Dinitrobenzen	Chemical with explosive properties/ środek chemiczny o właściwościach wybuchowych
Ethylene glycol/ Glikol etylenowy	Used in car radiators, in the synthesis of polyester resins, solvents, and explosive materials/ używany w katalizatorach samochodów; do syntezy żywic poliestrowych, roztworów i materiałów wybuchowych
Hexachlorophene/ Heksachlorofen	Antiseptic agent and disinfectant, used topically/ środek antyseptyczny i dezynfekcyjny, używany miejscowo
Iodoform/ Jodoform	Iodine derivative of methane; antiseptic agent used in endodontic treatment/ trójjodometan; środek antyseptyczny używany w leczeniu endodontycznym
Clioquinol/ Clioquinol	Antifungal and antiprotozoal agent; component of ointments, e.g. Lorinden C/ środek przeciwgrzybiczy i przeciwpierwotniakowy, składnik maści, np. Lorinden C
Methyl acetate/ Octan metylu	Used as a solvent and intermediate product in organic synthesis/ używany jako rozpuszczalnik i produkt pośredni w syntezie organicznej
Toluene/ Toluen	Used in the synthesis of explosive materials, pigments, fragrances, detergents, and aircraft fuels/ używany w syntezie materiałów wybuchowych, barwników, aromatów, detergentów oraz paliwa samolotowego

Tab. III. Chemical substances with potential toxic effect on the optic nerve.
Tab. III. Związki chemiczne o potencjalnie toksycznym wpływie na nerw wzrokowy.

Arsenide minerals/ Minerały arsenowe	Compounds of arsenide and metals; also used as semiconductors/ składowe arsenu i metali; używane jako półprzewodniki
Arsacetin/ Arsacetin	Organic arsenide mineral used in the therapy of syphilis/ organiczny minerał arsenowy używany w leczeniu kiły
Cobalt chloride/ Chlorek kobaltu	Added to fertilizers and animal fodders/ dodawany do nawozów i paszy dla zwierząt
Lead/ Ołów	Salts and oxides of this metal are highly toxic/ sole i tlenki tego metalu są silnie toksyczne
Thallium/ Tal	Highly toxic element; component of rodent poisons/ bardzo toksyczny składnik trucizn na gryzonie

Tab. IV. Metals and their allied products with potential toxic effect on the optic nerve.
Tab. IV. Metale i ich pochodne o potencjalnie toksycznym wpływie na nerw wzrokowy.

may be required in order to exclude the involvement of certain compounds.

Blood and urine tests play a crucial role in the evaluation of deficiency neuropathy. Tests should included complete blood count with WBC differential, and determining the serum levels of vitamin B_{12}, folic acid, and iron. Also, gastrological examination should be performedaimed at excluding gastrointestinal conditions associated with vitamin malabsorption.

Treatment

Causal factor elimination is the first step in the treatment of nutritional neuropathy. In some cases, the elimination of a harmful factor can resolve the condition. Another stage of the treatment should comprise implementating of a well-balanced, nutritious diet, rich in protein and green vegetables. The diet should be completed by the supplementation of vitamins, mostly B_1, B_{12}, and folic acid. Also, reducing alcohol consumption and cessation of smoking are recommended.

Appropriate preventive supplementation of vitamins is recommended in patients after bariatric surgery. Such patients should be administered 400 μg of folic acid (1–2 tablets), 300–500 μg of oral vitamin B_{12}, 400–800 IU of vitamin D, 1200–2000 mg of calcium citrate, 40–65 mg of iron, and 5000–10000 IU of vitamin A daily. The recommended dose of vitamin B_1 amounts to 50 mg/day in asymptomatic patients; this should be increased to 100 mg/day for 7–14 days in individuals with neurological symptoms, with subsequent decrease to 10 mg daily.

Implementation of the treatment, especially during the initial stages of neuropathy, is usually sufficient to improveme vision or even to restore full visual acuity. Starting the treatment prior to optic nerve atrophy is a prerequisite of treatment efficacy.

In the era of various alimentary pathologies, such as hunger and cachexia in poor and underdeveloped countries or excessive obesity and constant use of slimming diets in highly developed populations, nutritional dysfunction of the optic nerve constitutes a frequent problem and its prevalence will increase. We would like to highlight this problem by presenting the case of a young woman in whom chronic use of "water-based" diet resulted in anemia and bilateral nutritional optic neuropathy.

Case report

A 37-year-old patient was admitted to the Department of Ophthalmology of the University Clinical Hospital in Bialystok due to gradually progressing, bilateral impairment of visual acuity, with associated subjectively perceived color vision impairment, manifesting as hypersensitivity to bright colors. These problems persisted for about 3 months, and the patient was treated at the Regional Ophthalmology Outpatient Clinic for 2 months. As neither the ambulatory examination nor computed tomography

and magnetic resonance imaging of optic nerves and brain, as well as neurological and laryngological assessment revealed the reasons behind the ophthalmic problems reported by the patient, she was referred for further evaluation to the hospital.

Medical history taken on admission did not reveal any significant systemic conditions, injuries, or allergies. The patient did not use any prescription drugs on a regular basis. She declared occasional drinking of alcohol, and smoked about 10 cigarettes per day for several years. She had no family history of visual pathologies or other conditions.

On the second day of hospitalization, the patient admitted to using "water-based" diet for several months; this diet is based on drinking substantial amounts of water, about 5–8 liters per day, and reduced consumption of high-calorie foods. Vegetables and fruits constituted most of her meals, with only small amounts of meat.

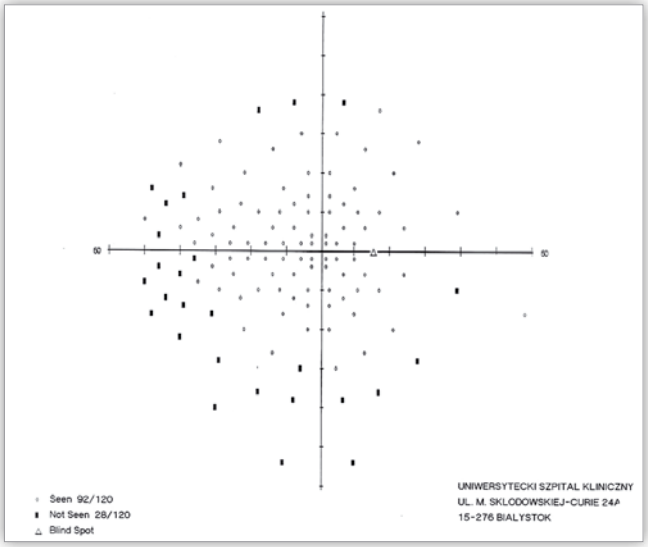

Fig. 3. Static perimetry of the right eye before treatment – peripheral scotomas.
Ryc. 3. Statyczne pole widzenia prawego oka przed leczeniem – mroczki obwodowe.

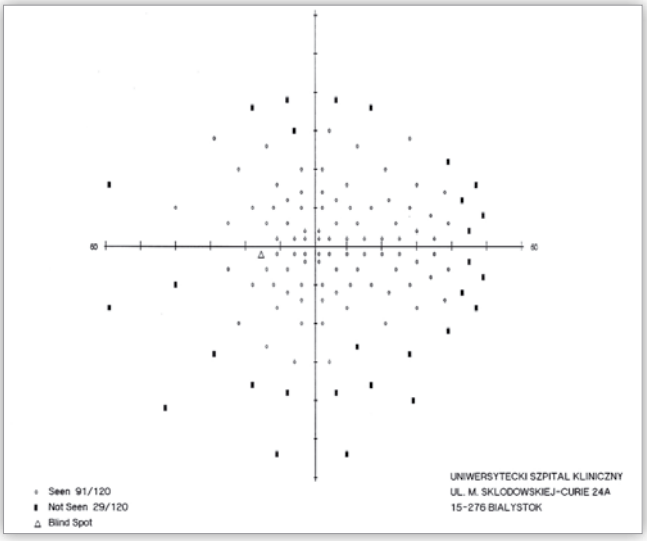

Fig. 4. Static perimetry of the left eye before treatment – peripheral scotomas.
Ryc. 4. Statyczne pole widzenia lewego oka przed leczeniem – mroczki obwodowe.

Ophthalmic examination performed on admission revealed bilateral decreased visual acuity for distant: OP – 5/25 cc -0.5 Dsph/-0.75 Dcyl ax 150° = 5/10, OL – 5/25 and near vision: OP – 1.0; OL – 3.0, normal color perception on the Ishihara plate, intraocular pressure in both eyes amounting to 15 mmHg, normal pupillary light reflex, and no abnormalities in the anterior segment and fundus of the eye.

Additional ophthalmic examination revealed abnormalities in visually evoked potentials (VEP) and perimetry. The VEP reading showed a decrease in the amplitude and normal latency of waves in both eyes. Peripheral scotomas were revealed in the both visual fields (Fig. 3, 4). Ultrasound examination, including Doppler ultrasonography, fluorescein angiography, and optical coherence tomography did not reveal any abnormalities.

The evaluation was completed with laboratory tests. Documented abnormalities included the signs of megalocytic anemia with slight thrombocytopenia (RBC: Red Blood Cell – 2.72 x 10^6/L ↓, HGB: Hemoglobin – 10.7 g/dL ↓, HCT (Hematocrit) – 30.9% ↓, MCV (Mean Corpuscular Volume) – 113.4fL ↑, PLT (Platelets) – 128 x $10^3/\mu$L ↓, anisocytosis ++; macrocytosis +++; hypochromia ++; ESR (Erythrocyte Sedymentation rate) – 3, slightly elevated blood triglycerides (236 mg/dL ↑), hyponatremia (134 mmol/L ↓), hypokaliemia (3.59 mmol/L ↓), decreased fraction of albumins in protein electrophoresis, and the presence of nitrates, bilirubin, and traces of ketone bodies on urinalysis. Upon hematologic consultation, serum folic acid and vitamin B_{12} concentrations were determined, revealing the deficiency of the latter (157 pg/mL, norm: 189–883 pg/mL). The patient underwent gastroscopy to verify the suspicion of malignant anemia, and gastric biopsy specimens were obtained for histopathological examination. The latter excluded type A chronic atrophic gastritis, and thus Addison-Biermer disease as a cause of megaloblastic anemia.

Continuous monitoring of arterial blood pressure (ABP), pulse, and body temperature revealed tachycardia, which persisted throughout the entire hospitalization; possibly, representing compensatory bodily response to anemia. The mean heart rate was 114 beats per min (104–133 beats per min), and the ABP amounted to 120–140/ 90–100 mmHg.

Due to the signs of vitamin B_{12} deficiency anemia, the treatment comprised properly balanced diet rich in protein and vitamin B complex, intramuscular injections of vitamin B_{12} (1000 μg), intravenous infusions of vitamin B_3, known to have neuroprotective effect and involved in erythropoiesis, as well as oral potassium preparations. Iron deficiency (18 ug/dL, norm: 40–140 ug/dL) was detected on hospitalization day 5, probably resulting from the crisis related to the increase in reticulocyte count, necessitating the use of oral iron preparations.

Marked improvement in bilateral visual acuity was documented after one week of treatment, along with the resolution of subjective ophthalmolic problems reported by the patient. Additionally, some laboratory parameters improved: RBC – 2.89 x $10^6/\mu$L, HGB – 11.1 g/dL, HCT – 33.3%, MCV – 115.0 fL, PLT – 175 x $10^3/\mu$L, K – 4.86 mmol/L, Na – 138 mmol/L, vitamin B_{12} – 35 ug/dL.

The patient was discharged home with full bilateral visual acuity for distant and near vision, normal color perception, and with no abnormalities of anterior and posterior eye segments. Perimetric examination revealed small peripheral defects

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in the left eye and normal visual field in the right eye (Fig. 5, 6). Discharge recommendations emphasized the necessity of rigorous sticking to a well balanced diet rich in protein and vitamins with additional oral supplementation of vitamin B_{12} and iron preparations. Furthermore, the patient was recommended to reduce the number of smoked cigarettes, and to refer regularly to the Hematology Outpatient Clinic for follow-up visits. At present, one year after discharge, the patient is followed by the Outpatient Ophthalmology Clinic of the University Clinical Hospital. Her general and local status is excellent.

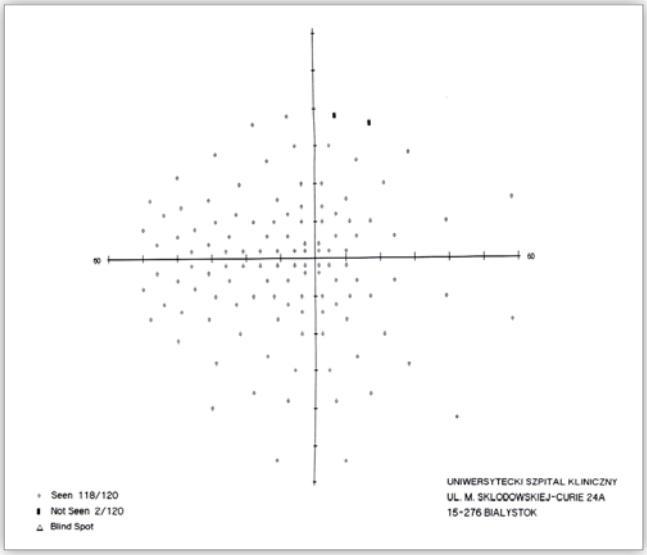

Fig. 5. Static perimetry of the right eye after treatment – normal field.
Ryc. 5. Statyczne pole widzenia prawego oka po leczeniu – prawidłowe pole.

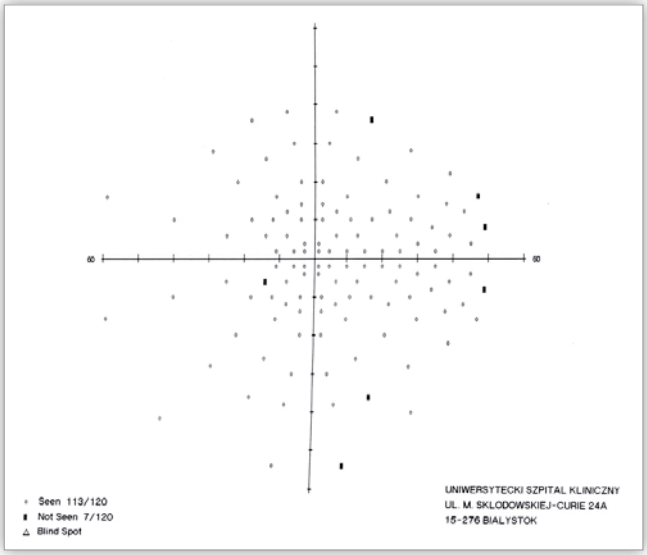

Fig. 6. Static perimetry of the left eye after treatment – small peripheral scotomas.
Ryc. 6. Statyczne pole widzenia lewego oka po leczeniu – małe mroczki obwodowe.

Discussion

Nutritional optic neuropathy can develop in otherwise healthy individual as a result of dietary "experiments" not supported by evidence-based dietetics. Due to their restrictive character, slimming diets aimed at losing possibly the largest amount of kilograms in a relatively short period of time may have serious health consequences. Appropriately devised process of slimming should be spread out over time and based on the rules of rational nutrition, providing the body with all the nutrients necessary for its functioning. Consequently, using slimming diets based on the elimination of certain types of food solely on one's discretion will result in deficiencies, which can manifest as more or less severe disorders and diseases, including optic neuropathy.

The same applies to individuals who due to various reasons, e.g. related to worldview, religion, or health, use various diets on a daily basis, including vegetarian diet. Some more radical forms of the latter allow for the consumption of only unprocessed fruits and vegetables. Used for longer time, such diets wreck tremendous havoc in the body as it is impossible to prepare nutritious meals based solely on the allowed food products. In such cases, the only solution is additional supplementation of deficient nutrients, mostly vitamins and microelements.

The presented case perfectly illustrates the idea that poorly balanced diet may have serious health consequences. This problem was previously highlighted by other authors who described similar cases of optic neuropathy resulting from folic acid and vitamin B complex deficiency (10, 11, 14, 15).

References:

1. Glaser JS: *Nutritional and toxic optic neuropathies*. In: Glaser JS, Ed. Neuro-ophthalmology. 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1999, 181–186.
2. Spinazzi M, De Lazzari F, Tavolato B, Angelini C, Manara R, Armani M: *Myelo-optico-neuropathy in cooper deficiency occuring after partial gastrectomy. Do small bowel bacterial overgrowth syndrome and occult zinc ingestion tip the balance?* J Neurol. 2007; 254: 1012–1017.
3. Orssaud C, Roche O, Dufier JL: *Nutritional optic neuropathies.* J Neurol Sci. 2007; 262; 158–164.
4. Plant GT, Dalmar AA: *Presumed nutritional optic neuropathy in Cuba, Tanzania, the Gambia and Somalia*. J Neurol Neurosurg Psychiatry. 2011; 82: 10.
5. Dalmarr AA, Hodson KE, Plant GT: *Epidemic optic neuropathy is evident in the Somalian population*. J Neuroophthalmol. 2011; 31: 127–130.
6. The Cuba Neuropathy Field Investigation Team: *Epidemic optic neuropathy in Cuba – clinical characterization and risk factors*. N Engl J Med. 1995; 333: 1176–1182.
7. Thomas PK, Plant GT, Baxter P, Bates C, Santiago Luis R: *An epidemic of optic neuropathy and painful sensory neuropathy in Cuba: clinical aspects*. J Neurol. 1995; 242: 629–638.
8. Cuban National Operative Group on Epidemic Neuropathy: *Epidemic neuropathy in Cuba*. Cuban Ministry of Health, Havana, Cuba, July 30, 1993.
9. Santiesteban-Freixas R, Pamias-Gonzalez E, Luis-Gonzales S, Serrano-Verdecia C, Gonzalez-Quevedo A, Alfaro-Capdegelle I, et al.: *Epidemic neuropathy: proposal and arguments to rename Strachan disease as Strachan and Madan disease*. Rev Neurol. 1997; 25: 1950–1956.
10. Hsu CT, Miller NR, Wray ML: *Optic neuropathy from folic acid deficiency without alcohol abuse*. Ophthalmologica. 2002; 216: 65–67.

11. de Silva P, Jayamanne G, Bolton R: *Folic acid deficiency optic neuropathy: a case report*. J Med Case Reports. 2008; 2: 299––301.
12. Chacko JG, Rodriguez CJ, Uwaydat SH: *Nutritional optic neuropathy status post bariatric surgery*. Neuro-Ophthalmol. 2012; 36: 165–167.
13. Becker DA, Balcer LJ, Galetta SL: *The neurological complications of nutritional deficiency following bariatric surgery*. J Obesity. 2012; ID 608534, 1–8.
14. Nightingale LM, Paviour DC: *Nutritional optic and peripheral neuropathy: a case report*. Cases J. 2009; 2: 7762–7765.
15. Golnik KC, Schaible ER: *Folate-responsive optic neuropathy.* J. Neuroophthalmol. 1994; 14: 163–169.

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