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Nutrition assessment of patients with AMD

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ABSTRACT

The aim of the study was to assess the nutritional habits of AMD patients from the Szczecin region. Age-related macular degeneration (AMD) is a chronic, progressive eye disease that results in loss of central vision. The study was conducted in the Ophthalmology Clinic of the Independent Public Teaching Hospital No. 2, Pomeranian Medical University (PUM) in Szczecin among 46 subjects. The interview method was used using the author's questionnaire, which contained questions about age, body weight and height, sex, composition of the three-day menu and the form of AMD. Insufficient intake of numerous nutrients (with lutein and zeaxanthin among them) has been shown, which may accelerate the progression of the disease. In addition, patients were underweight, which may increase the risk of protein-energy malnutrition that may affect the development of AMD.

***Keywords*:** age-related macular degeneration, carotenoids, EFAs, vitamins, bioelements, AMD

1. INTRODUCTION

Age Related Macular Degeneration (AMD) is a chronic, progressive disease. The macula lutea, which is the central part of the retina, is responsible for visual acuity and enables the perception of details of viewed objects, as well as recognition of colours and a sense of contrast [1].

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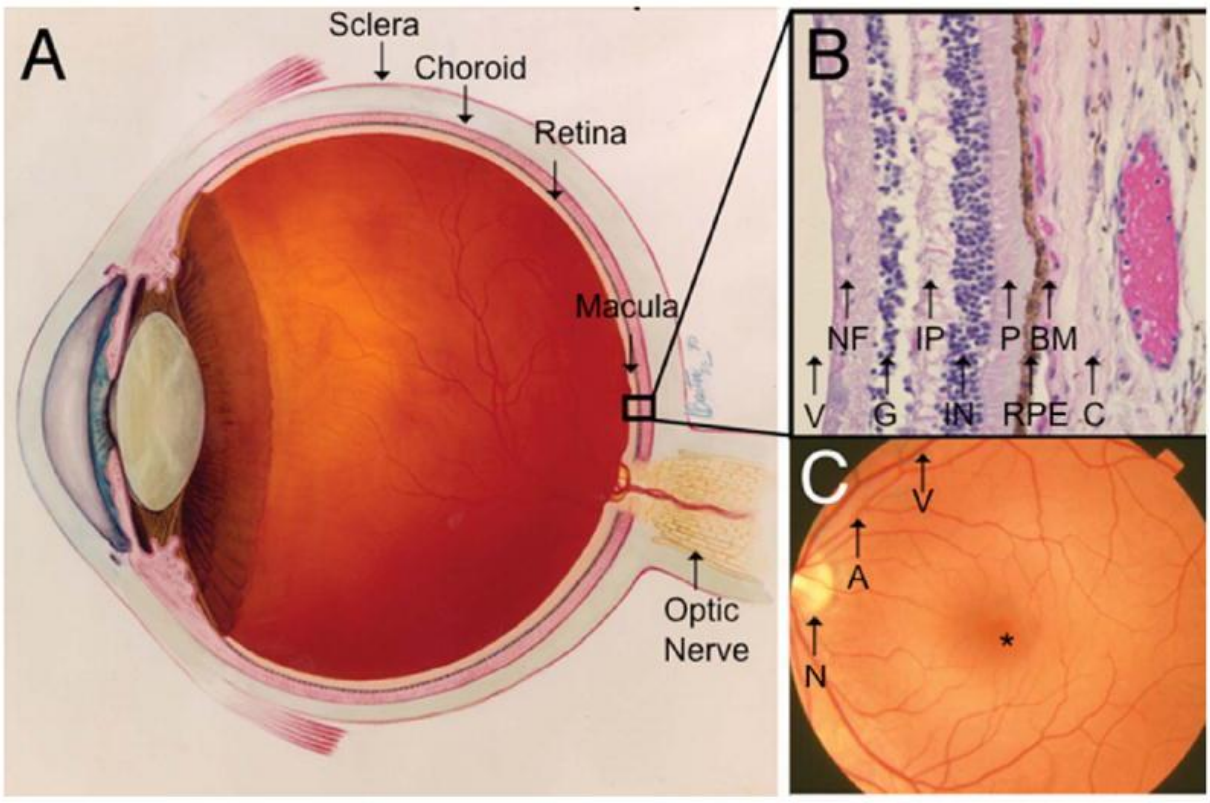

Figure 1. A) A cross-section of the eye that shows the retina and adjacent anatomical structures. B) Histological cross-section of the retina, which includes: vitreous body (V), nerve fibre layer (NF), ganglion cell layer (G), inner plexus layer (IP), inner nuclear layer (IN), photoreceptor layer (P), retinal pigment epithelium (RPE), Bruch's membrane (BM), choroid (C). C) A fundoscopic image that shows the macula lutea located between the vascular arcades, including the retinal artery (A) and vein (V) with the faeva centralis (*), as well as the optic nerve (N) [2].

As a result of the progression of the disease, which affects people over 50, there is a loss of central vision and, as a consequence, loss of sight [1].

Photoreceptors are photosensitive macular receptors. To function properly, they must be constantly reconstructed by the cells nourishing them that need a lot of oxygen and energy. With age, the metabolic function is disturbed, which results in the deposition of a brown pigment (lipofuscin) in the macula, which is a product of incomplete degradation of the external segments of photoreceptors. As a result of the deficiency of nutrients supporting the natural defence system of the macula, free oxygen radicals and toxic lipid metabolites are produced. Thus, oxidative stress is generated, and toxic substances accumulate in the macula and impair the function of photoreceptors and nourishing tissues.

Drusen are formed, which are punctual deposits under the pigment epithelium. Drusen blocks the supply of blood and nutrients. New blood vessels are formed, which may result in the destruction of other eye structures. Photoreceptors and the nutrients nourishing the macula die, resulting in loss of sight [1].

Figure 2. Image seen by a healthy person (on the right) and a person suffering from AMD (on the left) [3].

2. RESULTS

The number of women and men in both groups was: 8 and 38, respectively. The study reported the wet form of AMD in 41 patients and the dry one in 5 patients (which may result from collecting a group with the wet form of AMD for the research programme being implemented in the Ophthalmology Clinic of the Independent Public Teaching Hospital No. 2, Pomeranian Medical University (PUM) in Szczecin). None of the subjects systematically took dietary supplements. The interview method was used using the author's questionnaire, which contained questions about age, body weight and height, sex, composition of the three-day menu and the form of AMD.

The nutritional status of study participants was determined by calculating the BMI, then the results obtained were compared with the normal levels for older people. Next, the obtained results were compared with the normal levels for people over 65 years of age determined by the Committee on Diet and Health (1989) [4]. The reason for choosing these normal levels was the fact that they take into account the older age of patients; moreover, they are most appropriate for the assessment of the Polish population, as stated by Babiarczyk and Turbiarz [5]. The average BMI in the group of men aged 66-75 was 19.2 kg/m^2 and for those >75 years old – 18.5 kg/m^2, while for women aged 66-75 it was 18.4 kg/m^2 and for those >75 years – 18.7 kg/m^2 (Table 1).

The groups of people surveyed showed too low body weight – underweight compared to the commonly accepted BMI levels in the range 20-24.9, and especially at a BMI value of 25-30 kg/m^2 for people over 65 years of age (Table 1). The BMI value of 25-30 kg/m^2 is a normal level accepted by the Committee on Diet and Health and is increasingly used in Poland due to the lowest risk of death in elderly patients; moreover, it concerns 39.9%, while in the case of the BMI 20- 24.9 only 25% of people above 65 years of age [5].

Table 1. BMI of women and men aged 66-75 and> 75 with AMD (K – women, M – men).

	Range	Arithmetic mean
M 66-75	18.1-22.3	19.2
K 66-75	17.9-19.1	18.4
M >75	17.7-19.3	18.5
K >75	18.1-19.3	18.7

Underweight was the result of no energy demand coverage and incorrect supply of carbohydrates, proteins and fats in the diet of the subjects. The energy normal level fulfilment for men and women ranged from 56 to 84% (Table 2). The coverage of protein, fat and carbohydrate demand was 84-97%, 64-74% and 42-100%, respectively (Table 2).

Table 2. Nutritional interview results. Average nutrient intake by men and women, normal level values and percentage of normal level fulfilment (K – women, M – men).

Nutrient	Age range	Normal level	Mean +/- SD	% Normal level fulfilment
Energy value (kcal)	M 66-75	2160	1491 +/- 503.09 SD	69
	>75	2050	1262 +/- 370.41 SD	61
	K 66-75	1890	1596 +/- 267.79 SD[4]	84
	>75	1840	1031 +/- 168.75 SD[4]	56
Protein (g)	M 66-75	63	59.4 +/- 23.16 SD[3]	94
	>75	63	44 +/- 17 SD[3]	70
	K 66-75	56	53.9 +/- 12 SD	96
	>75	56	54 +/- 21 SD	96
Fat (g)	M 66-75	84	62.1 +/- 32.81 SD	74
	>75	80	51.4 +/- 18.04 SD	64
	K 66-75	74	49.5 +/- 6.25 SD	67
	>75	72	48.9 +/- 3.57 SD	68
Polyunsaturated fatty acids: DHA and EPA (mg)	M 66-75	190-650	955 +/- 7.98 SD	
	>75	190-650	600 +/- 5 SD	
	K 66-75	190-650	622 +/- 2 SD	
	>75	190-650	600 +/- 2 SD	

Total carbohydrates (g)	M 66-75 >75 K 66-75 >75	288 270 250 242	188.2 +/- 61.17 SD 167.2 +/- 57.75 SD[2] 249.8 +/- 70.84 SD[4] 102.8 +/- 20.35 SD[24]	65 62 100 42
Sugars (g)	M 66-75 >75 K 66-75 >75	216 205 189 184	42.4 +/- 18.86 SD 44 +/- 21.66 SD 64.2 +/- 22.84 SD 37 +/- 1.57 SD	20 21 34 20
Sucrose (g)	M 66-75 >75 K 66-75 >75		33.3 +/- 19.22 SD[1] 34 +/- 24 SD[2] 52.1 +/- 11 SD[14] 3 +/- 1 SD[24]	
Lactose (g)	M 66-75 >75 K 66-75 >75		9.1 +/- 7.09 SD 10 +/- 9 SD 12.1 +/- 5 SD 2 +/- 2 SD	
Fibre (g)	M 66-75 >75 K 66-75 >75	25 25 25 25	15.8 +/- 6.01 SD[3] 12 +/- 6 SD[3] 18.7 +/- 3 SD[4] 10 +/- 3 SD[4]	63 48 75 40
Sodium (mg)	M 66-75 >75 K 66-75 >75	1300 1200 1300 1200	1598 +/- 1024.68 SD 1539 +/- 747 SD 2183.4 +/- 972 SD 1372 +/- 213 SD	123 128 168 114
Potassium (mg)	M 66-75 >75 K 66-75 >75	4700 4700 4700 4700	2088 +/- 854.27 SD[3] 1506 +/- 574 SD[3] 1981 +/- 159 SD 1341 +/- 873 SD	44 32 42 29
Calcium (mg)	M 66-75 >75 K 66-75 >75	1200 1200 1200 1200	659 +/- 353.36 SD 602 +/- 364 SD 724,8 +/- 405 SD 326 +/- 118 SD	55 51 60 27

Phosphorus (mg)	M 66-75 >75 K 66-75 >75	700 700 700 700	1026 +/- 419 SD 756 +/- 300 SD 977.3 +/- 181 SD 814 +/- 372 SD	146 108 140 116
Magnesium (mg)	M 66-75 >75 K 66-75 >75	420 420 320 320	225 +/- 111.65 SD 157 +/- 59 SD 216.8 +/- 35 SD 167 +/- 93 SD	54 37 68 53
Iron (mg)	M 66-75 >75 K 66-75 >75	10 10 10 10	7.9 +/- 2.98 SD[3] 6 +/- 2 SD[3] 8.1 +/- 2 SD 5 +/- 2 SD	79 60 81 50
Zinc (mg)	M 66-75 >75 K 66-75 >75	11 11 8 8	7.77 +/- 3.04 SD[3] 6 +/- 2 SD[3] 7.6 +/- 1 SD 6 +/- 3 SD	71 55 95 75
Copper (mg)	M 66-75 >75 K 66-75 >75	0.9 0.9 0.9 0.9	0.87 +/- 0.39 SD 1 +/- 0 SD 0.9 +/- 0 SD 1 +/- 0 SD	97 111 100 111
Manganese (mg)	M 66-75 >75 K 66-75 >75	2.3 2.3 1.8 1.8	3.13 +/- 1.62 SD[3] 2 +/- 1 SD[3] 3.4 +/- 1 SD 2 +/- 1 SD	136 87 189 111
Selenium (µg)	M 66-75 >75 K 66-75 >75	55 55 55 55	25.5 +/- 9.96 SD 28 +/- 10 SD 26.8 +/- 7 SD 24 +/- 11 SD	46 51 49 44
Vitamin A (µg)	M 66-75 >75 K 66-75 >75	900 900 700 700	754 +/- 601.62 SD 516 +/- 358 SD 780.7 +/- 362 SD[4] 285 +/- 86 SD[4]	84 57 112 41

Vitamin B_1 (mg)	M 66-75 >75 K 66-75 >75	1.3 1.3 1.1 1.1	0.79 +/- 0.31 SD[3] 1 +/- 0 SD[3] 0.8 +/- 0 SD 1 +/- 0 SD	61 77 73 91
Vitamin B_2 (mg)	M 66-75 >75 K 66-75 >75	1.3 1.3 1.1 1.1	1.19 +/- 0.45 SD 1 +/- 0 SD 1.2 +/- 0 SD 1 +/- 0 SD	92 77 109 91
Vitamin PP (mg)	M 66-75 >75 K 66-75 >75	16 16 14 14	11.09 +/- 6.52 SD[3] 5 +/- 2 SD[23] 9.8 +/- 4 SD 10 +/- 5 SD[2]	69 31 70 71
Vitamin B_6 (mg)	M 66-75 >75 K 66-75 >75	1.7 1.7 1.5 1.5	1.37 +/- 0.59 SD[3] 1 +/- 0 SD[3] 1.4 +/- 1 SD 1 +/- 1 SD	81 59 93 67
Vitamin B_{12} (mg)	M 66-75 >75 K 66-75 >75	2.4 2.4 2.4 2.4	2.42 +/- 1.14 SD 2 +/- 1 SD 2.2 +/- 1 SD 2 +/- 1 SD	101 83 92 83
Vitamin C (mg)	M 66-75 >75 K 66-75 >75	90 90 75 75	46.1 +/- 44.97 SD 30 +/- 37 SD[2] 66.4 +/- 45 SD[4] 4 +/- 6 SD[24]	51 33 89 5
Vitamin D (µg)	M 66-75 >75 K 66-75 >75	15 15 15 15	1.88 +/- 2.23 SD 1 +/- 1 SD 0.9 +/- 0 SD 3 +/- 2 SD	13 7 6 20
Vitamin E (mg)	M 66-75 >75 K 66-75 >75	10 10 8 8	6.49 +/- 5.07 SD[3] 4 +/- 3 SD[3] 5.7 +/- 3 SD[4] 3 +/- 1 SD[4]	65 40 71 38

Folic acid (µg)	M 66-75 >75 K 66-75 >75	400 400 400 400	138.9 +/- 68.18 SD[3] 95 +/- 46 SD[3] 139 +/- 36 SD[4] 58 +/- 22 SD[4]	35 24 35 15
Lutein (mg)	M 66-75 >75 K 66-75 >75	10 10 10 10	1.39 +/- 0.46 SD 1 +/- 0 SD 1.3 +/- 1 SD 1 +/- 0 SD	14 10 13 10
Zeaxanthin (mg)	M 66-75 >75 K 66-75 >75	2 2 2 2	1.4 +/- 6.28 SD 1.6 +/- 5 SD 1.2 +/- 2 SD 1.4 +/- 9 SD	70 80 60 70
Lycopene (mg)	M 66-75 >75 K 66-75 >75	35 35 35 35	19.05 +/- 6.59 SD 19 +/- 7 SD 20.8 +/- 6 SD 20 +/- 4 SD	54 54 59 57
Cholesterol (mg)	M 66-75 >75 K 66-75 >75	300 300 300 300	187 +/- 123.42 SD 186 +/- 134 SD 172 +/- 81 SD 148 +/- 43 SD	

[1] – statistically significant differences between men and women 66-75 years, [2] –statistically significant differences between men and women >75 years, [3] – statistically significant differences between men 66-75 and over 75 years, [4] – statistically significant differences between women 66-75 and over 75 years.

It was shown that sucrose intake (p = 0.039) in women and men in the age group 66-75 years differed statistically significantly. It can therefore be concluded that women consumed more sucrose than men. In the case of the other nutrients, no statistically significant differences in their intake between men and women of the same age, i.e. >66 years of age, were found.

However, statistically significant differences appeared between respondents in the age group >75 years in terms of consumed carbohydrates (p = 0.022), sucrose (p = 0.008), as well as niacin (0.039) and vitamin C (0.007). The average values of carbohydrate, sucrose and vitamin C intake were higher among men, whereas niacin in women. This means that men consumed more carbohydrates, sucrose and vitamin C than women, and women more niacin than men. In terms of other nutrients, no statistically significant differences were found (p > 0.05). The intake of food ingredients by men aged 66-75 was also compared with the group over 75 years of age. It was proved that for protein, fibre, potassium, iron, zinc, manganese,

vitamin E, thiamine, niacin, B6 and folates, the result of the Mann Whitney U test was statistically significant at $p < 0.05$. Therefore, statistically significant differences were found between the intake by men aged 66-75 and those over 75 years of age of: protein ($p = 0.045$), fibre ($p = 0.026$), potassium ($p = 0.024$), iron ($p = 0.010$), zinc ($p = 0.036$), manganese ($p = 0.031$), vitamin E ($p = 0.048$), thiamin ($p = 0.039$), niacin ($p = 0.000$), vitamin B6 ($p = 0.004$) and folates ($p = 0.024$). The average content of these nutrients in the diet was higher in the group of younger men. In terms of other nutrients, no statistically significant differences were found ($p > 0.05$).

When comparing the intake by women aged 66-75 with the group over 75 years of age, it was shown that the average energy value of the diet, as well as that of carbohydrates, sucrose, lactose, fibre, vitamins A, E and C and folates, differed statistically at $p < 0.05$. The average content of these nutrients was higher in the diets of women aged 66-75. This means that younger women consumed more of these nutrients than women over 75 years of age. In terms of other nutrients, no statistically significant differences were found ($p > 0.05$).

3. DISCUSSION

Many studies conducted in patients with age-related macular degeneration show that a properly balanced diet in terms of nutrients, i.e. lutein, zeaxanthin, lycopene, vitamins A, C, E, minerals: iron, zinc, selenium and copper, and fatty acids: EPA and DHA, affects the delay of AMD progression [6].

One of the most reliable clinical studies assessing the impact of diet on AMD prevention was the Age-Related Eye Disease Study (AREDS 1 and its continuation AREDS 2) [7, 8]. In both programmes, patients' diets were enriched with:

- AREDS 1: vitamin C – 500 mg, vitamin E – 400 IU, beta-carotene – 15 mg, zinc (as oxide) – 25 and 80 mg, copper (as oxide) – 2 mg,
- AREDS 2: beta-carotene was abandoned in favour of lutein – 10 mg, zeaxanthin – 2 mg, vitamin C – 500 mg, vitamin E – 400 IU, copper (as oxide) – 2 mg, EPA – 650 mg, DHA – 350 mg.

The AREDS 1 study reported that using only antioxidants slowed disease progression by 17%, zinc by 21%, and zinc and antioxidants by 25%. It turned out that such therapy in the subjects reduces the risk of wet AMD by 25%. However, beta-carotene led to lung cancer in smokers. This is due to the fact that in contact with free radicals contained in tobacco smoke, as well as at high oxygen pressure (comparable to atmospheric one), the activity of some carotenoids changes from antioxidant to pro-oxidant. Therefore, in the AREDS 2 study, beta-carotene was abandoned in favour of lutein and zeaxanthin, and supplementation with eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) was added, while the remaining nutrients remained unchanged. A 30% reduction in the risk of wet AMD has been reported [7].

It has been proved that eating fish more often than twice a week reduces the risk of both early and later AMD (by about 38%) [9].

In 2014, an experimental study on rats was performed to check the effect of a high fructose diet on the development of pathological changes in age-related macular degeneration. Initially, the animals were treated with a laser to induce AMD. In a study using indocyanine green angiography, it has been shown that the use of a high fructose diet resulted in a greater

number of pathological changes indicating macular degeneration than the standard diet. There has been a clear increase in body weight when using this diet. In addition, subretinal neovascularisation has occurred earlier. A relative lutein deficiency within the macula has also been demonstrated [10].

According to the opposite view, nutrition does not affect the appearance of age-related macular degeneration. Causes of the disease should be sought in: family history of AMD, genes, cardiovascular diseases (hypertension, coronary artery disease, atherosclerosis), smoking, hypercholesterolaemia, diabetes, light skin and iris colour, increased exposure to UV radiation [6].

In the study conducted at the Teaching Hospital No. 2 in people suffering from AMD, an increased risk of protein-energy malnutrition was found, which resulted in weight loss and deterioration of well-being reported by patients (including lethargy and depression). It can be assumed that malnutrition could be one of the factors accelerating the development of AMD. As a result of the deterioration of vision, a decrease in patients' mobility was observed, which forced families to take care of them [11].

The analysis of menus also showed that the normal values for zeaxanthin and lycopene were not met. It should be noted that the normal values for these carotenoids only provide an estimate of intake. Lutein and zeaxanthin are pigments found in the macula and are a natural filter of blue light. They play a protective role for photoreceptors by preventing oxidative stress-induced apoptosis. Lycopene has the ability to take unpaired electrons of lutein and zeaxanthin after their reaction with free radicals. Deficiencies of other nutrients with antioxidant activity: vitamins C, E, zinc and selenium, has also been demonstrated. Vitamin C can affect the functioning of the lens, because it is secreted into the aqueous humour, where its concentration is higher than in other body fluids.

Vitamin E is found in the cell membranes of photoreceptors and in the pigment epithelium, playing a protective role against oxygen free radicals. Deficiencies can accelerate the ageing process of the eyes. In turn, in the retina and choroid is the highest concentration of zinc in the body, which may indicate its indispensability. In addition, zinc acts as a coenzyme for antioxidant enzymes. Its deficiency can lead to myopia.

Considerable deficiencies in intake have also been reported for vitamin A. It is often called an eye-epithelial-growth vitamin. It plays a significant role in biochemical transformations, because it participates in the visual cycle. In the form of a precursor, it is necessary for re-synthesis of rhodopsin, which conditions the mesopic vision. In addition, vitamin A improves epithelial function and protects the conjunctiva from drying out. However, too much supply is inadvisable with macular degeneration as it causes the formation of bis-retinoid (A2E). On the other hand, the deficiency results in structural and functional changes of the conjunctiva, cornea and retina of the eye, as well as mucous membranes and skin (especially around the outlets of the hair follicles) [1, 12, 13].

The analysis of the menus shows that the subjects had a normal supply of polyunsaturated fatty acids: EPA and DHA. Omega 3 fatty acids are responsible for eye hydration and improve the quality of tears. EPA inhibits angiogenesis and inflammation in the retinal pigment epithelium (RPE), while DHA accelerates the elimination of lipofuscin from RPE. In addition, there was adequate copper and manganese supply, and high lutein intake. In the available literature, no data was found on the adverse effects of excess lutein on the human body. However, it is known that copper and manganese exhibit antioxidant activity only when supplied in accordance with normal values [12].

The research of other authors shows that people diagnosed with macular degeneration had more knowledge about lutein-rich products compared to healthy people. Unfortunately, this did not translate into lutein intake [14, 15]. In addition, people with AMD had a reduced level of physical activity associated with progressive amblyopia [11].

Although our own research showed statistically significant differences, they concerned relatively small groups of patients, especially the oldest ones, which is why we treat the results as those requiring confirmation in a larger number of patients. The need for further research is also indicated by conflicting views in the literature on the value of food ingredients in preventing or inhibiting the progression of macular degeneration.

In view of the conflicting views on the value of food ingredients in preventing or inhibiting the progression of macular degeneration and a relatively small group of patients, there is a need for further studies assessing the correlation of diet and nutrition of patients with the clinical course of AMD.

4. CONCLUSIONS

Patient diets have shown significant deficiencies in many nutrients, including lycopene and zeaxanthin, which can aggravate disease progression, especially in people with dry AMD.

A large percentage of patients with low body weight was found in the group of respondents, which increases the risk of protein-energy malnutrition that may affect the development of AMD.

People with AMD had a reduced level of physical activity, which was directly related to progressive amblyopia.

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