ДOI: https://doi.org/10.25276/2410-1257-2020-4-17-19
УДК 617.735

Внезапная потеря зрения у больного сахарным диабетом с липемией сетчатки и атрофией зрительного нерва

Мехмет Омер Кырыштыоглу[1], Гамзе Укан Гюндюз[2], Озгур Ялчинбаир[3], Онер Гелишкен[4]

[1]*Резидент. Отделение офтальмологии. Медицинский факультет университета Улудаг, Бурса, Турция*
[2]*Руководитель. Отделение офтальмологии. Медицинский факультет университета Улудаг, Бурса, Турция*
[3]*Ассоциированный профессор. Отделение офтальмологии. Медицинский факультет университета Улудаг, Бурса, Турция*
[4]*Профессор. Больница Озел Джимер, Бурса, Турция*

Sudden Vision Loss in a Diabetic Patient with Lipemia Retinalis and Optic Atrophy

Mehmet Omer Kiristioglu[1], Gamze Ucan Gunduz[2], Ozgur Yalcinbayir[3], Oner Gelisken[4]

[1]*Resident. Department of Ophthalmology. Uludag University School of Medicine, Bursa, Turkey*
[2]*Instructor. Department of Ophthalmology. Uludag University School of Medicine, Bursa, Turkey*
[3]*Associate Professor. Department of Ophthalmology. Uludag University School of Medicine, Bursa, Turkey*
[4] *Professor. OzelJimerHastanesi, Bursa, Turkey*

РЕФЕРАТ

Липемия сетчатки – редкое, как правило, бессимптомное осложнение в результате повышенного уровня сывороточных триглицеридов. Представленный случай описывает первый известный в литературе случай липемии сетчатки с атрофией зрительного нерва.

Клинический случай. У 41-летнего мужчины с сахарным диабетом II типа в анамнезе, эпилепсией, потерей слуха, гипертонией и нарушением мозгового кровообращения наблюдалось снижение зрения правого глаза. У пациента была двусторонняя липемия сетчатки и бледность диска зрительного нерва с правой стороны, обусловленная, вероятно, длительной микрососудистой дисфункцией.

Вывод. В условиях липемии сетчатки и гипертриглицеридемии у данного пациента развилась односторонняя атрофия зрительного нерва и потеря зрения. Потеря зрения была стойкой и не ассоциировалась с самой липемией сетчатки. Пациент прошел лечение гиполипидемической терапией, но после липемической стадии зрение пациента не изменилось.

Ключевые слова: *липемия сетчатки, атрофия зрительного нерва, гиперлипидемия.* ■

Точка зрения. Восток – Запад. 2020;4:17-19.

ABSTRACT

Lipemia retinalis is a rare, generally asymptomatic complication of elevated serum triglycerides. This case describesfirst known case of lipemia retinalis with optic atrophy in the literature.

Case Report. A 41-year-old male with a history of type II diabetes mellitus, epilepsy, hearing loss, hypertension and cerebrovascular accident presented with decreased vision in his right eye. He had bilateral lipemia retinalis and right sided optic nerve pallor in the setting of probable long lasting microvascular dysfunction.

Conclusion. In the setting of lipemia retinalis and hypertriglyceridemia, the current patient developed unilateral optic atrophy and vision loss. Vision loss was permenant and not associtated with lipemia retinalis itself. He was treated with lipid lowering therapy but after lipemic phase, patient's vision did not change.

Key words: *lipemia retinalis, optic atrophy, hyperlipidemia.* ■

Point of View. East – West. 2020;4:17-19.

INTRODUCTION

Lipemia retinalis (LR) is a rare clinical entity that can be seen in both primary (familial) and secondary (systemic disease) hyperlipidemia syndromes due to high blood triglyceride levels. Scattering of the light ray by the plasma chylomicrons in the retinal blood vasculature causes this ocular finding [1]. Lipemia retinalis was first defined by Heyl in 1880 as an ocular lipemia [2].

Fading of red reflex of retinal vasculature and salmon colored choroidal reflex because of changes in the choroidal vessels are characteristics of this clinical entity. Earliest findings can be detected in the peripheral retina. With the increase of blood triglyceride lev-

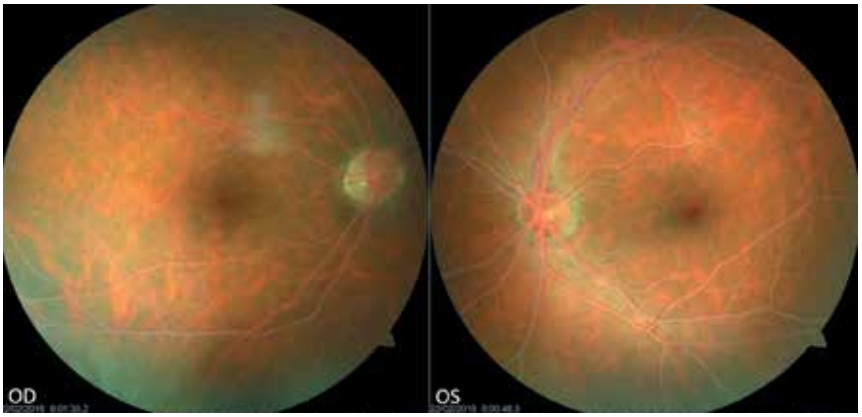

Fig. 1. Fundus images at the time of presentation. Note that there was optic nerve pallor in the right eye. Both eye had fading color of retinal vasculature salmon colored choroidal reflex

Fig. 2. Fundus images one month after presentation. Note that there was optic nerve pallor in the right eye. Since lipemic state had regressed, color of retinal vasculature and choroid reflex returned to relatively normal appearance

els, findings progress into the posterior pole [3]. Despite this striking retinal and choroidal changes, LR is rather an incidental finding due to not causing visual loss usually.

An acute increase in level of triglyceride might be asymptomatic in the beginning and at this stage, LR can be seen as a vital clinical finding in a relatively healthy patient. Hyperlipidemia without hypertriglyceridemia does not cause lipemia. Lipemia retinalis is only seen in patients with primary hyperlipidemia associated with high chylomicron levels (ie type I, III, IV and V). Secondary hyperlipidemias with increased triglyceride levels can be seen in patients with uncontrolled diabetes mellitus, systemic lupus erythematosus, hypothyroidism, nephrotic syndrome, obesity, alcoholism, renal insufficiency and biliary obstruction [4].

Case report. A 41 year old patient with no history of known ophthalmologic disease presented with sudden painless vision loss in his right eye to the emergency department. In his medical history, patient had type II diabetes mellitus, epilepsy, hearing loss, history of cerebrovascular accident and hypertension. In presentation, his vital signs were normal. In his ophthalmological examination, he had relative afferent pupillary defect in his right eye. Both direct and indirect light reflexes were positive but direct light reflex of the right eye was sluggish. He had no pain with eye movements. His visual acuity was hand motion in right eye and his best corrected visual acuity (BCVA) was 0.8 in left eye via Snellen chart by decimal. Slit lamp examination revealed no specific findings in the anterior segment except relative fading of conjunctival vessels. Intraocular pressure was 11 mmHg in the both eyes. Fundus examination showed creamy color in the peripheral retina and posterior pole, choroidal reflexes were salmon colored and grade 1 hypertensive retinopathy in both eyes *(Fig. 1)*. Right optic disk was pale with defined margin, while left one had defined margin with healthy disc appearance. He had no lipid exudation or diabetic retinopathy. Fundus fluorescein angiography revealed no retinal vasculature occlusion.

According to his detailed medical history and records, he had epilepsy and treated with carbamazepine 1000 mg/day po and clonazepam 0.5 mg bid po since he was 7 year old. His family history was unremarkable. He had been operated on biliary tract due to obstruction. He had cerebrovascular accident when he was 27 years old. After the accident, he had experienced visual and auditory hallucinations. His psychiatric evaluation revealed mild mental retardation. There was ischemic gliotic foci in his cranial magnetic resonance imaging, and electroencephalography revealed abnormal theta waves. He had hearing loss in his left ear since childhood but he had hearing loss in his right ear since 30 years old.

His blood pressure was normal but his blood glucose level was 399 mg/dL. Blood lipid parameters couldn't be measured because of lipemic serum with extremely high levels. We emergently referred the patient to the internal medicine department to lower blood lipid levels and metabolic regulation to prevent further end organ damage. After systemic evaluation, patient had been discharged from emergency room after proper metabolic regulations.

One month after presentation his visual acuity was unchanged. Slit lamp examination revealed no specific findings in the anterior segment. Fundus examination revealed relatively normal retinal vasculature relatively normal in color with grade 1 hypertensive retinopathy findings *(Fig. 2)*. Lipemia retinalis had totally regressed but optic disc pallor was still existing in his right eye. In optical coherence tomography, there was relative atrophy of the inner retinal layer in macula. In his control blood lipid profile tests, total cholesterol level was 306 mg/dL, HDL cho-

lesterol level was 12 mg/dL, direct LDL cholesterol level was 118 mg/dL and triglyceride was 1531 mg/dL. His CBC test was unremarkable, erithrocyte sedimentation rate was 17 mm/hour, and his thyroid function tests were within normal limits. His coagulation parameters and blood homocystein level was in the normal range. He had glucosuria without ketonuria in his urine analysis.

DISCUSSION

Lipemia retinalis is a rare clinical manifestation. It can be seen primarily in familial lipid disorders or secondarily to systemic metabolic disease. It is often a short lasting and asymptomatic manifestation so it is hard to detect clinically. According to a few reported cases, retinal vascular disease can be associated with LR [5]. In this case, since patient had presented with acute painless monoocular vision loss, we had to rule out retinal vascular diseases like central retinal artery occlusion. But it is not always easy, due to paleness of the retinal vasculature. Bilateral paleness of the both arterial and venous vasculature and fundus fluoresceine angiography findings led us to differential diagnosis.

Uncontrolled diabetes mellitus and high serum lipids might be underlying reasons of cerebrovascular accident, hearing loss, mild mental retardation and even psychiatric symptoms due to microvascular pathologies. The patient was uncooperative and his metabolic state was highly uncontrolled.In his biochemical tests, LDL cholesterol and apolipoprotein B was normal, HDL was low and HbA1c was very high. With his negative family history of primary lipid disorders and his blood tests' led us to diagnosis of secondary disease like uncontrolled diabetes mellitus. But unfortunately, we couldnot perform serum lipoprotein electrophoresis since patient didn't approve further blood tests.

Wolfram syndrome is a rare condition that affect many parts of the body. Diabetes insipidus, diabetes mellitus, hearing loss and optic atrophy are the pillar of this syndrome. But in this syndrome, optic atrophy is generally bilateral unlike our patient, and appears around age of eleven [6]. In 70% of patients with Wolfram syndrome have diabetes insipidus with some levels of other pituitary hormones insufficiencies [6]. But in this case, diabetes insipidus wasnot noted. We wanted to rule out Wolfram syndrome with genetic testing but the patient did not approved the genetic testing.

In previous studies, visual function alterations and electroretinography changes were reversible in case of short lasting hypertriglyceridemia [7, 8]. But in cases of long lasting hypertriglyceridemia irreversible changes could happen because of sustained retinal ischemia.

Yanko et al. [9] studied an experimentally induced hyperlipoproteinemia with high fat and high cholesterol diet with ablation of the thyroid tissue in Rhesus monkeys. They researched the effect of hyperlipoproteinemia on retina and optic nerve. According to this study, one monkey had bilateral temporal pallor of optic discs with ophthalmological findings and two of four monkeys' postmortem examination showed segmental lesions where the neuroglial pattern of the optic nerve was disrupted in cross sections of the optic nerve. There were also an almost total absence of axons which was associated with diminution of myelin and presence of enlarged reactive astrocytes and microglial proliferation but the central retinal arteries and veins were found potent in this sections. This lesions were strongly resemble those found in chronic ischemic optic neuropathy. Hayreh et al [10] reported that similar pathological characteristics of the optic nerve atrophy could be found after experimental occlusion posterior ciliary artery in monkeys. Lieberman et al [11] indicated that embolic occlusion of pial or pial derived arterioles might similarly produce segmental infarction of the optic nerve. In our patient, optic atrophy might be due to previous anterior ischemic optic neuropathy associated with uncontrolled diabetes, hypertension, or hyperlipidemia.

Since lipemia retinalis is a harbinger of uncontrolled metabolic state, urgent regulation of blood glucose levels, lipid lowering therapy with dietary and lifestyle changes are vital to prevent cardiovascular morbidity/mortality and retinal ischemia.

REFERENCES

1. Ahrends EH Jr, Kunkel HG. The stabilization of serum lipid emulsions by serum phospholipids. J Exp Med. 1949;90:409-24.
2. Heyl AG. Intra-Ocular Lipæmia. Trans Am Ophthalmol Soc. 1880;3:54-66.
3. Anderson DR, Davis EM. Retina and optic nerve after posterior ciliary artery occlusion. An experimental study in squirrel monkeys. Arch Ophthalmol. 1974;92:422-6.
4. Brown M, Goldstein J. The hyperlipoproteinemias and other disorders of lipid metabolism. In: Braunwald E, Isselbacher KJ, Petersdorf RG, Nilson JD, Martin JB, Fauci AS, editors. Harrison Principles of Internal Medicine, 11 thed. NY-MGH Int.
5. Nagra PK, Ho AC, Dugan JD Jr. Lipemia retinalis associated with branch retinal vein occlusion. Am J Ophthalmol. 2003 Apr;135(4):539-42.
6. Barrett TG, Bundey SE, Macleod AF. Neurodegeneration and diabetes: UK nationwide study of Wolfram (DIDMOAD) syndromeLancet. 1995 Dec 2;346(8988):1458-63. PubMed PMID: 7490992.
7. Ong JM, Zorapapel NG, Aoki AM, et al. Impaired electroretinogram (ERG) response in apolipoprotein E deficient mice. Curr Eye Res. 2003;27:15-24.
8. Lu CK, Chen SJ, Niu DM, et al. Electrophysiological Changes in Lipaemia Retinalis. Am J Ophthalmol. 2005;139:1142-45
9. Yanko L, Michaelson IC, Rosenmann E, Ivri M, Lutsky I. Effects of experimental hyperlipoproteinaemia on the retina and optic nerve in rhesus monkeys. Br J Ophthalmol. 1983 Jan;67(1):32-6.
10. Hayreh SS, Baines JAB. Occlusion of the posterior ciliary artery. I. Effects on the optic nerve head. Br J Ophthalmol. 1972;56: 754-64.
11. Lieberman MF, Shahi A. Embolic ischemic optic neuropathy. Am J Ophthalmol. 1978;86:206-10.