(27)

Optical coherence tomography in imaging of macular diseases

Optyczna koherentna tomografia w obrazowaniu chorób plamki

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Summary: OCT (opitcal coherence tomography), is a diagnostic method that enables the analysis of the retinal structures by means of high-resolution tomographic cross-sections of the retina. Whereas fluorescein and indocyanine angiography allow visualization of the retinal epithelium layer and chorioretinal vessels, OCT may help in diagnosing and monitoring the condition of many internal retinal layers. Ultrasound B-mode examinations have a resolution of about 150 μm while OCT provides a resolution of 10 μm. OCT makes it possible to detect and measure morphological changes, retinal thickness, retinal volume, thickness of retinal nerve fiber layer and various parameters of the optic disc. We use OCT in the analysis of the retinal structures in various pathologies such as macular holes and pseudo-holes, epiretinal membranes, macular edemas of various origins, including vasooclusive disease and diabetic macular edema, lesions of vitreoretinal interface and vitreoretinal traction, serous and hemorrhagic detachments of the retina and of pigment epithelium, age related macular degeneration, diabetic retinopathy, glaucoma. OCT is an examination that is fast, sensitive, reproducible, non-invasive, non-contact and easy to perform and interpret for a retinologist.
The aim of this article is to present OCT principles and techniques as well as OCT interpretation and images of most common retinal diseases: age related macular degeneration – dry and wet form, retinal angiomatous proliferation (RAP), central serous chorioretinopathy, epiretinal membranes, macular holes, diabetic retinopathy.

Słowa kluczowe: optyczna koherentna tomografia, technika, interpretacja, zwyrodnienie plamki żółtej związane z wiekiem – sucha postać, mokra postać, proliferacja naczyniowa siatkówki (RAP), retinopatia surowicza środkowa, błony nasiatkówkowe, otwory w plamce, retinopatia cukrzycowa.

Key words: optical coherence tomography, technique, interpretation, age related macular degeneration – dry, wet form, retinal angiomatous proliferation (RAP), central serous chorioretinopathy, epiretinal membranes, macular holes, diabetic retinopathy.

Optical coherence tomography (OCT), is a universal diagnostic method enabling high resolution tomographic cross-sections of the retina and detailed analysis of its structure. Fluorescein and indocyanine angiography allow visualization of the retinal epithelium layer and chorioretinal vessels. OCT makes it possible to spatially image the morphology of retinal layers, the border between the retina and the vitreous body and between the retina and the choroid. USG in presentation B yields the resolution of 150 μm while OCT approximately 10 μm. OCT is a repeatable examination. Thanks to the available database, the following control tomograms can be compared.

OCT examination of the posterior pole is especially useful in diagnostics and treatment of:

- macular holes and pseudo-holes,
- vitreoretinal traction,
- macular retinoschisis,
- epiretinal membranes,
- macular edemas of various origins, including vasooclusive disease and diabetic macular edema,
- serous and hemorrhagic detachments of the retina and of pigment epithelium in the macula,
- age related macular degeneration,
- diabetic retinopathy,
- severe or chronic epitheliopathy,
- subretinal or intraretinal neovascularization,
- retinal atrophy,
- central serous retinopathy,
- glaucoma – evaluation of the retinal nerve fiber layer and the optic disc.

OCT enables monitoring of:

- retinal morphology and its changes,
- thickness of the retina in particular areas and points,
- volume of the retina in particular areas and points,
- thickness of the retinal nerve fiber layer,
- parameters of the optic disc.

The aim of this survey is to outline the OCT techniques and present the most common diseases of the posterior poll of the eyeball based on the characteristic of their OCT images.

1. OCT techniques

SOCT Copernicus HR is one of the devices based on the method of spectral optical tomography, which allows for obtaining three dimensional images of human eye. The partly coherent light beam is reflected by the internal structures of the retina and then subjected to interferometric analysis. It allows for obtaining high-quality tomographic images. Superluminescent diode

ISSN 0023-2157 Index 362646

is the source of 855 nm light. The device does not have a reference mirror. The images are characterized by high axial resolution (dependant on the type of used source of light) – 3 μm in the tissue and transverse (number of A scans per each image in projection B) – typical 18 μm. The depth of the scan amounts to 2 mm, the width is between 4-10 mm (1.2).

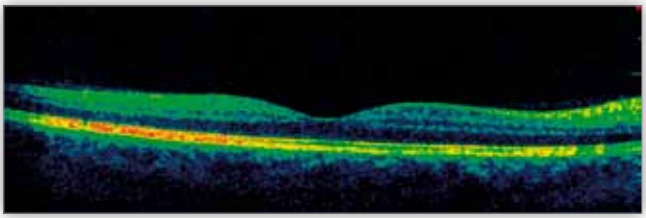
In Stratus OCT device the wavelength of 830 nm is emitted by a superluminescent diode and it is aimed at optical beam splitter. One of the beams reaches the examined tissue where it is partially absorbed, reflected and dispersed. The second beam is reflected by reference mirror, which moves backwards and forwards. The light of both beams on their way back cause interference, which is then digitalized and analyzed by a computer, which forms a single A scan. The scanning point moves longitudinally creating the image of B scan.

In the Stratus OCT device the transverse resolution (number of A scans in B scan), is 20 μm. The longitudinal resolution is the exponent of the light wavelength of the luminescent diode and it is 10 μm for this device (1.2).

2. Basics of OCT interpretation

The basics of OCT interpretation are the changes in the reflectivity of different retinal layers, which depend on the type of pathology. The following pathologies are characterized by high reflectivity (red or white areas on color OCT scans):

- dye aggregates,
- retinal pigment epithelium overgrowth,
- subretinal blood,
- scars,
- neovascularisation foci,
- hard exudates,
- physiological nerve fibers and retinal pigment epithelium-choriocapillaries complex (Fig. 1).



Ryc. 1. Normal macula in OCT.
Fig. 1. Prawidłowa plamka w OCT.

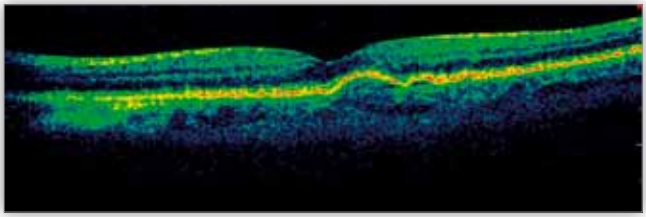
The following pathologies are characterized by low reflectivity:

- retinal pigment epithelium (RPE) atrophy,
- intraretinal fluid spaces – extended, cyst-like, under the retina and RPE,
- physiologically for the structures distributed vertically such as photoreceptors or nucleated layers of the retina (2).

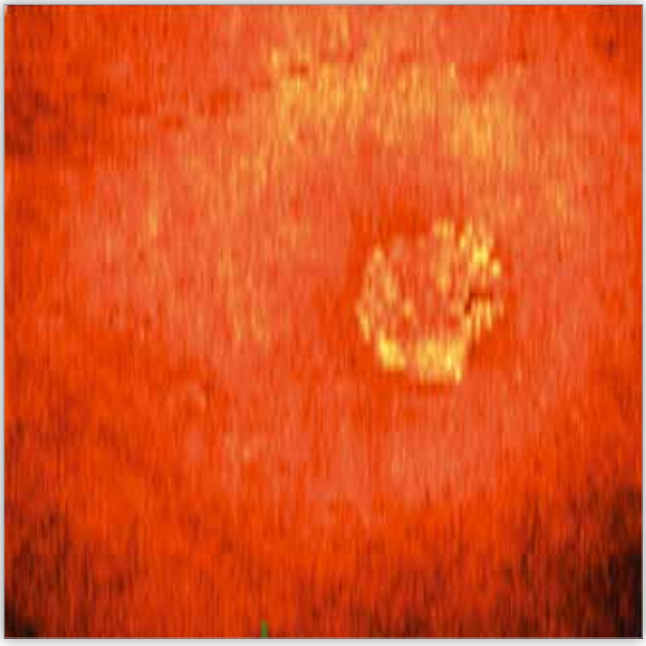
3. Practical use of OCT – diagnostics of dry form of age related macular disease (AMD)

Age related macular disease is a disease of central retina – macula, an area characterized by high density of photoreceptors. Macula is responsible for sharp vision and the perception of contrasting stimuli. AMD affects the retinal pigment epithelium, Bruch's membrane and choriocapillaries of the choroid causing their destruction. As indicated by the name of the disease, the risk of the development of the disease is age-related – it increases above 50 years of age. The disease mainly affects the developed countries. The incidence of the disease increases rapidly between 70-80 years of age. At that age it affects nearly 30% of the population. Other factors, which are conducive to developing AMD are female sex, smoking tobacco (oxidative stress), atherosclerosis, arterial hypertension, obesity, low physical activity, increased exposition to UV light, diet rich in fats and carbohydrates, insufficient supply of microelements. It is also known that AMD is inherited in a multifactorial way. Monozygotic twins are particularly susceptible to the disease. There are two clinical types of AMD: the mild one – dry (90% of cases), and the more severe wet one (10% of cases), which can result in the loss of central vision.

Soft and hard druses are typical for dry AMD. They are formed as a result of accumulation of the products of retinal pigment epithelium metabolism with insufficient efficiency of purification mechanism. The progression of this sort of changes results in the development of atrophy or transformation into exudative AMD. In OCT, druses deform and thicken the line of retinal pigment epithelium with local detachments (Fig. 2). Merging soft druses form drusenoids.

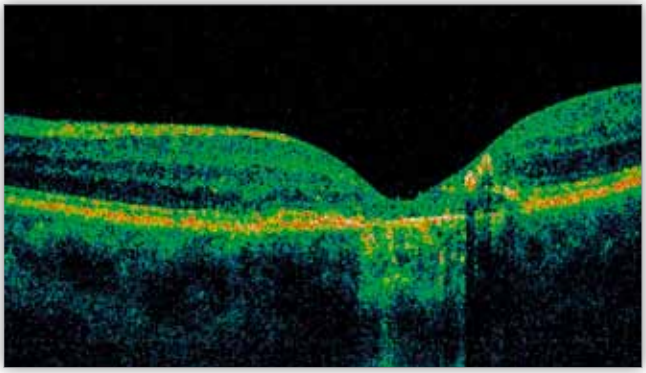


Ryc. 2. Dry AMD in OCT – drusen.
Fig. 2. AMD – postać sucha – w OCT – druzy.



Ryc. 3. Color picture – dry AMD, macular atrophy.
Fig. 3. Kolorowe zdjęcie – postać sucha atrofii plamki.

Progressing atrophy of the retinal pigment epithelium, the external layers of the retina and choriocapillaries results in the



Ryc. 4. Scan B – macular atrophy with reduction in retinal thickness and increase in the reflectivity of retinal pigment.
Fig. 4. Skan B – atrofia plamki z redukcją grubości siatkówki i wzrostem reflektywności nabłonka barwnikowego.

development of so called geographic atrophy of the macula, which is characterized by a certain image of the fundus of the eye. (Fig. 3). In OCT it is seen as a decrease in the thickness of the retina, increase in the reflectivity of the retinal pigment epithelium and increased penetration of light waves in the choroid (Fig. 4).

In summary, dry type AMD in OCT imaging is characterized by:

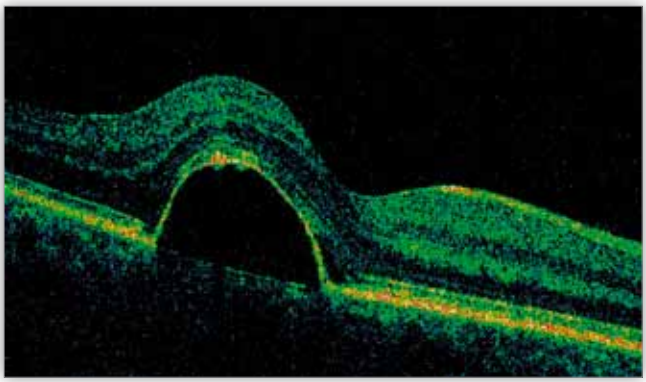
- druses increasing the thickness of RPE layer and deforming its line – seen as hyperreflectivity in OCT,
- damages in RPE, areas with atrophy of RPE, external layers of the retina and choriocapillaries – seen in OCT as decreasing the thickness of the retina, intensified choroidal reflection (1.3.4).

3.1. Diagnostics of exudative type of age related macular disease

Choroidal neovascularisation plays the key role in the pathogenesis of this AMD form. The deposits of RPE basilemma and lipid concretions of Bruch's membrane are conducive to the hypoxia of the retina, which is the most important signal for the production of vascular new growth mediators such as vascular endothelial growth factor (VEGF) and proinflammatory cytokines. They initiate the proliferation and migration of endothelial cells of the choriocapillaries. New inferior vessels cross the Bruch's membrane thanks to proteases, which are activated by the listed factors. The vessels locate horizontally against RPE (neovascularisation type I), in the subretinal space (neovascularisation type II), or in both those locations (mixed type). They resemble capillary tubes and they then differentiate into arterioles and venules. Type I of neovascularisation is typically multifocal. Patients do not report severe clinical symptoms and the changes are difficult to visualize in angiography and they correspond to hidden CNV. Type II of neovascularisation, located subretinally, can be mono- or multifocal. Pathological vessels, which are not tight, move underneath the retina and cause severe vision symptoms. This type of changes correlates with classical choroidal neovascularisation in angiographic imaging.

Following are the typical components of exudative AMD observed in OCT: fluid under the retina and RPE, retinal edema and subretinal hemorrhage. Subretinal blood can pass through the internal layers of the retina and accumulate on its surface or even in the vitreous body. On the border of the serous elevation of RPE exudates are often observed.

Serous pigment epithelial detachment (PED), is characterized by the presence of optically clean space between the retinal pigment epithelium and choriocapillaries with clearly indicated angle between those layers (Fig. 5). It can be monofocal or multifocal but also flat and widespread. Hemorrhagic retinal pigment epithelial detachment cannot be penetrated by OCT waves because of blood. Thus the evaluation of deeply located structures is impossible.



Ryc. 5. Serous pigment epithelium detachment.
Fig. 5. Surowicze odwarstwienie nabłonka barwnikowego.

In the course of exudative AMD subretinal fluid is a very important indicator of the disease activity. Subretinal fluid spaces are typically flat, often widespread and located at the border of subretinal membrane. The following indicator of the activity of choroidal neovascularisation (CNV), is the edema of the retina with optically empty, hyporeflective, intraretinal fluid spaces. Cyst-like, intraretinal edema can result in losses of retinal walls or layers.

Classical CNV (type II), in OCT images is characterized by the presence of hyperreflective focus comprising the RPE-choriocapillaries complex localized in front of the retinal pigment epithelium. The neovascular membrane has a focal and extensive character. Active CNV focus is usually accompanied by edema, thickened retina in the membrane area and subretinal fluid. The line of epithelium in the neovascular membrane area is deformed and thickened (Fig. 6-11).

The hidden form of CNV (type I, does not cross the pigment epithelium). It is characterized by the presence of expressly deformed RPE line with PED foci, the presence of subretinal fluid and possible retinal edema (Fig. 12).

In summary, in exudative AMD in OCT images one can observe:

- hyperreflective deformations and thickened RPE-choriocapillaries line in the CNV site,
- the activity of the disease is confirmed by: hyporeflective fluid spaces under the retina, under the retinal pigment epithelium, subretinal edema in advanced stages of the disease a hyperreflective subretinal scar may occur (Fig. 13. 14),
- OCT enables differentiating between the classic type CNV localized under the retina and the hidden type, which does not cross the RPE line (5.6)

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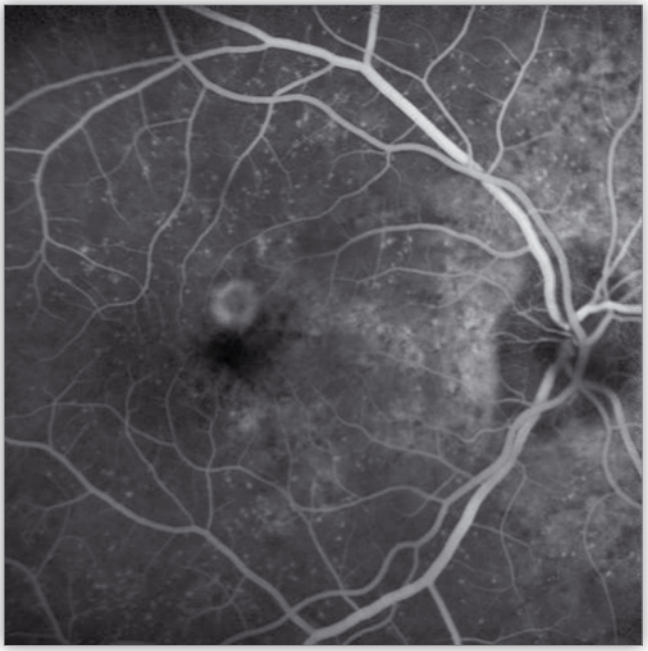
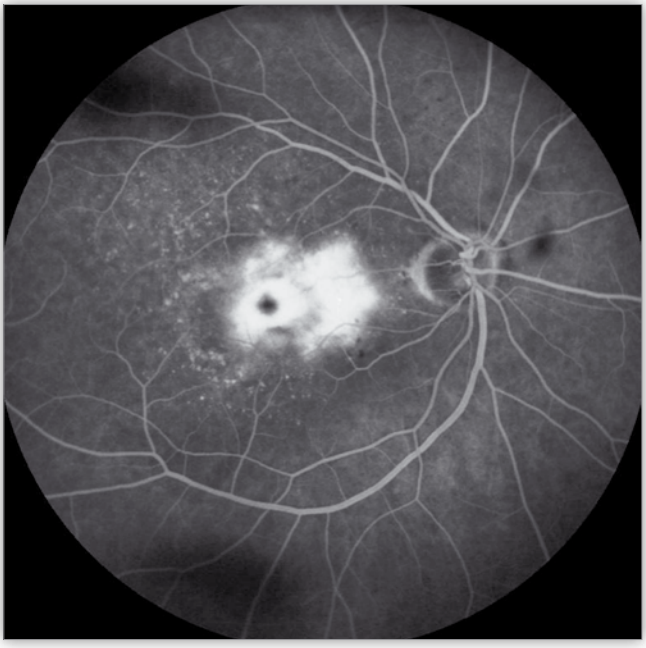
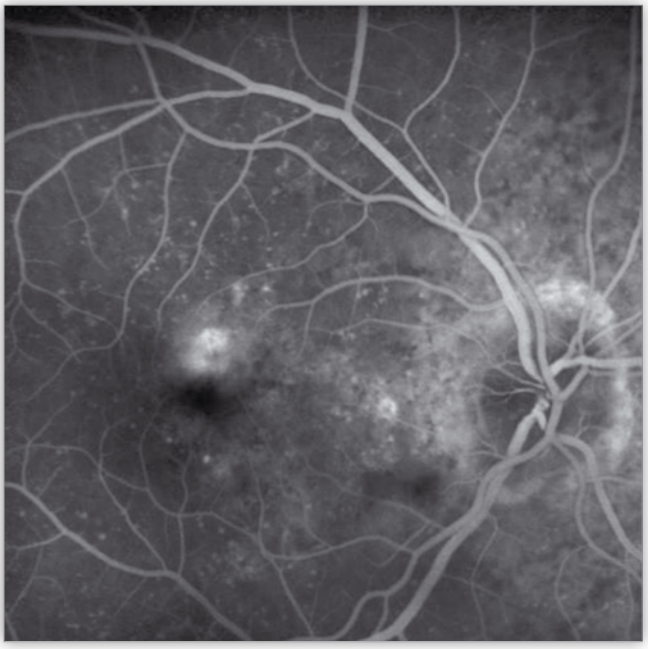
Ryc. 6. Classic wet AMD in fluorescein angiography.
Fig. 6. Klasyczne AMD o postaci wysiękowej w angiografii fluoresceinowej.



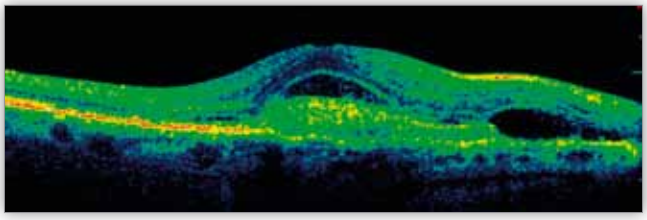
Ryc. 9. Juxtafoveolar focus of CNV in fluorescein angiography.
Fig. 9. Okołodołkowe ognisko CNV w angiografii fluoresceinowej.



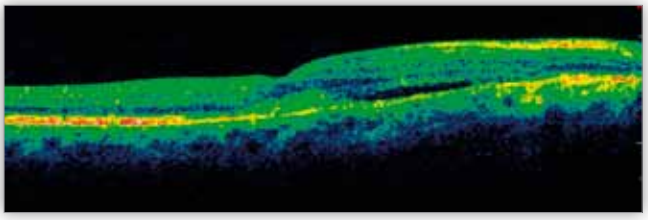
Ryc. 7. Classic wet AMD – late phase of fluorescein angiography, intensive hyperfluorescence of subretinal membrane.
Fig. 7. Klasyczne AMD o postaci wysiękowej – późna faza angiografii fluoresceinowej, intensywna hiperfluorescencja błony podsiatkówkowej.



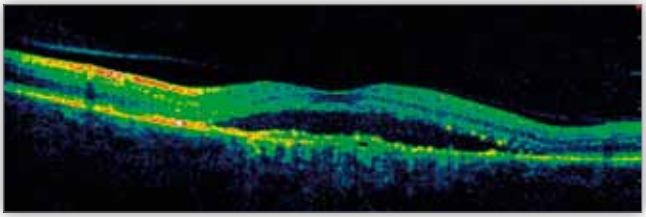
Ryc. 10. Juxtafoveolar focus of CNV –late phase of fluorescein angiography, intensive hyperfluorescence of subretinal membrane.
Fig. 10. Okołodołkowe ognisko CNV – późna faza angiografii fluoresceinowej, intensywna hiperfluorescencja błony podsiatkówkowej.



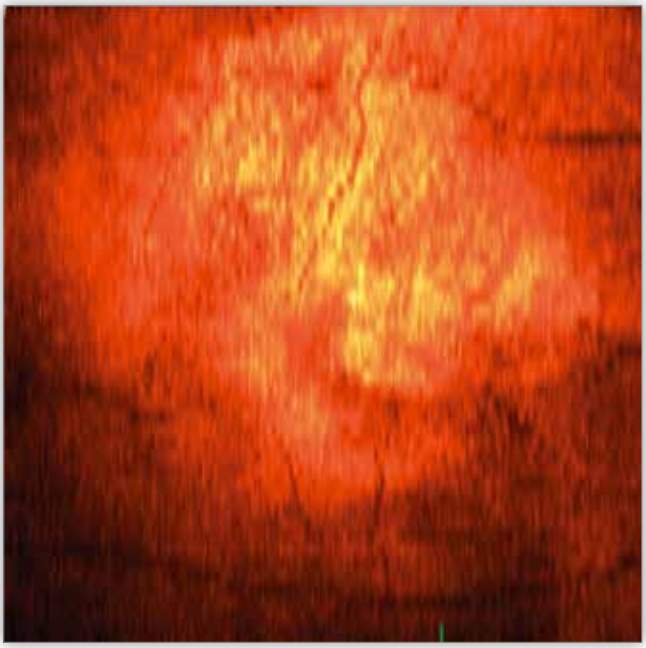
Ryc. 8. Classic wet AMD – increased reflectivity CNV with subretinal fluid in OCT.
Fig. 8. Klasyczne AMD o postaci wysiękowej – wzmożona refleksyjność CNV z płynem podsiatkówkowym w OCT.



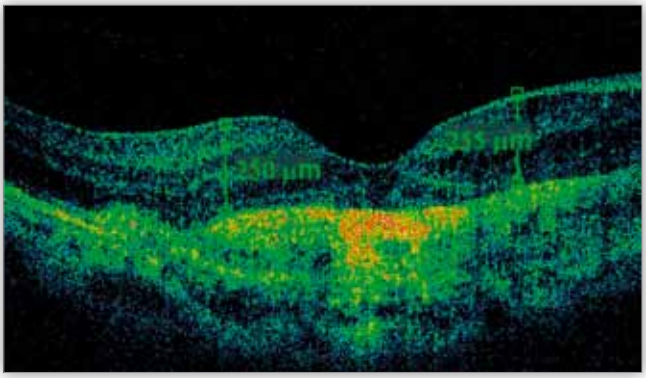
Ryc. 11. Juxtafoveolar hyperreflective focus of CNV with subretinal fluid in OCT.
Fig. 11. Okołodołkowe hiperreflektywne ognisko CNV z płynem podsiatkówkowym w OCT.



Ryc. 12. Occult wet AMD – subretinal fluid, distortion of RPE.
Fig. 12. Ukryte AMD o postaci wysiękowej– płyn pod siatkówką, zniekształcenie linii RPE.



Ryc. 13. Color picture of macula – subretinal scar.
Fig. 13. Kolorowe zdjęcie plamki – blizna podsiatkówkowa.

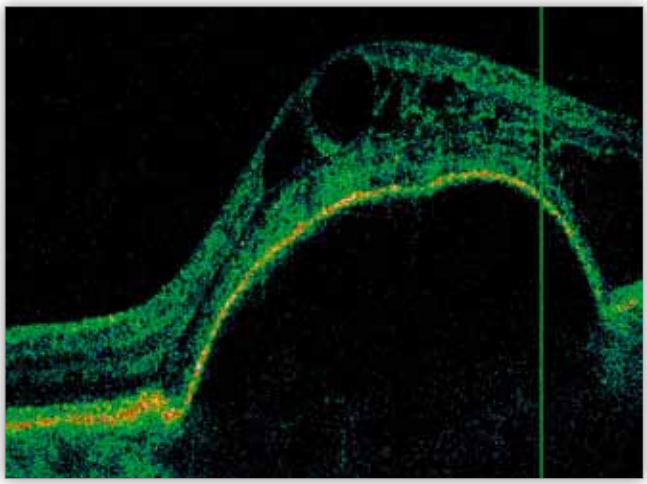


Ryc. 14. Hyperreflective subretinal scar in OCT – small retinal edema, without subretinal fluid.
Fig. 14. Hiperreflektywna blizna podsiatkówkowa w OCT – niewielki obrzęk siatkówki, bez płynu podsiatkówkowego.

3.2 Retinal angiomatous proliferation (RAP) in OCT

This type of exudative maculopathy originally develops in the retina. Intraretinal vascular proliferations are created, hemorrhages and intraretinal edema (stadium I) occur. The RAP progression results in the increase of the intraretinal edema, the creation of foci of retinal pigment epithelium and subretinal fluid spaces detachment as well as hemorrhages and exudates (stadium II). In this stadium a characteristic OCT image occurs. Stadium III is characterized by retinochoroidal anastomoses (undetectable by OCT).

At the fundus of the eye in RAP there is a grey elevation with microhemorrhages and exudates on the border. OCT examination usually shows high detachment level of retinal pigment epithelium with the subretinal fluid on the border. The retina is characterized by advanced edema with hyporeflective, cyst-like fluid spaces (Fig. 15). In RPA treatment vitreous injection of VEGF-A inhibitors is usually used, however relapses and scar formation at the final stages are often observed (7).



Ryc. 15. Retinal angiomatous proliferation in OCT – high PED elevation, retinal edema.
Fig. 15. Siatkówkowa proliferacja naczyniakowata w OCT – wysoka elewacja PED, obrzęk siatkówki.

4. OCT in retinal membranes diagnostics

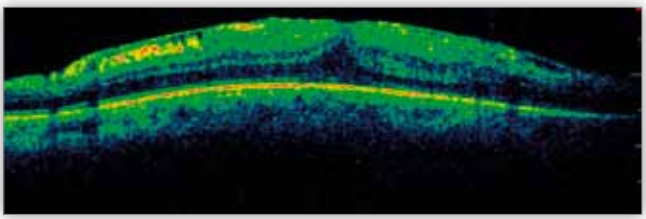
Epiretinal membrane – macular pucker (synonyms: preretinal gliosis, cellophane maculopathy), develops on the vitreoretinal border. It is composed of proliferating glia cells. These cells reach the retinal surface through holes in the internal border membrane. It is suspected that these holes are created during the posterior detachment of the vitreous body in the macular area.

Idiopathic membranes are usually observed in healthy people, who are above 50 years of age. They occur bilaterally in 10% of cases. The membranes can also be created secondarily after surgery of retinal detachment, photocoagulation, cryotherapy and as a result of vascular diseases of the retina and intraocular inflammation damage of the organ of vision. In the initial stadium of cellophane maculopathy minor vision distortion can occur in patients. The vision sharpness is correct or slightly impaired. In ophthalmoscopy the epiretinal membrane is transparent, thin and the macula has an irregular reflex. With time the epiretinal membrane gets thicker. It shrinks and creases are formed on the macula. Major vision distortions occur and the sharpness of vision is impaired. A change of vascular pattern in the macula is visible in ophthalmoscopy. It is confirmed in fluorescein angiography.

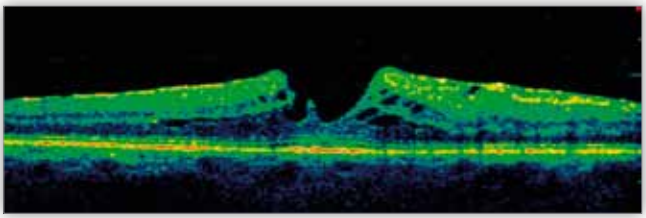
In OCT hyperreflective epiretinal membranes with traction with inner layers of the retina are observed in OCT. Tractions with a membrane and vitreous body are often observed too. Distortion of the depression outline and increasing the thickness

ISSN 0023-2157 Index 362646

of the retina in the foveola to more than 200 μm as a result of extended intraretinal edema (Fig. 16) are typical. A loss of retinal layers can occur until the formation of pseudo-holes (Fig. 17). In advanced stadiums of the disease surgical intervention is required – vitreoretinal operation with the removal of epiretinal membrane (4,8).



Ryc. 16. Hyperreflective epiretinal membrane, diffuse retinal edema.
Fig. 16. Hiperreflektywna błona nasiatkówkowa, rozlany obrzęk siatkówki.



Ryc. 17. Epiretinal membrane with lamellar hole and retinal edema in OCT.
Fig. 17. Błona nasiatkówkowa z otworem warstwowym plamki i obrzękiem siatkówki w OCT.

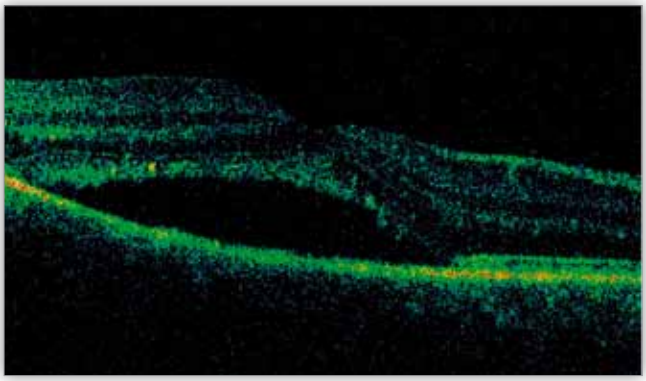
5. Central serous chorioretinopathy in OCT

This disease is characterized by usually unilateral, limited detachment of the sensory retina with a possible associated pigment epithelium detachment. In mild cases the disease retreats spontaneously. It typically affects young or middle-aged people, mostly men with the A type personality.

In the pathogenesis of serous central chorioretinopathy the incorrect functioning of retinal pigment epithelium as the external blood-retina barrier but also increased permeability of the choroidal vascularisation. Distorted vision and moderate impairment of sharpness are among typical symptoms. At the fundus of the eye an oval, grey protrusion of sensory retina is visible. In fluorescein angiography at the location of the leak from the side of retinal pigment epithelium there is a visible focus of increasing hyperfluorescence that can be characterized as "ink blot or chimney" smoke with contrasted fluid subretinal space. In 80% of cases the disease is mild, the leak retreats within six months and the vision improves. In around 20% of cases chorioretinopathy lasts up to 12 months and it is associated with permanent changes in the retinal pigment epithelium. Even if the sharpness of vision gets back to its original condition, minor discrete vision distortions are still possible. The disease may be relapsing in nature. In some cases the changes may occur on both sides.

In OCT an elevation of the retinal depression and the outline of the depression are typically preserved. Under the retina, in the depression, there is a hyporeflective, sometimes extensive, subretinal fluid space (Fig. 18). In the area of retinal pigment epithelium damage and leak, the focus of the retinal pigment epithelium detachment is observed. The regression of central serous chorioretinopathy in OCT is characterized by absorption of subretinal fluid and disappearance of hyporeflective subretinal fluid space. Distortion of RPE line can be a permanent defect as well as losses and thickening of the epithelium (in fluorescein angiography, granular hyperfluorescence within the area of former detachment of the retina).

OCT is the basic, non-invasive tool enabling fast diagnosing of central serous choriopathy and monitoring its development and treatment (4.9-11).



Ryc. 18. Serous retinal detachment.
Fig. 18. Surowicze odwarstwienie siatkówki.

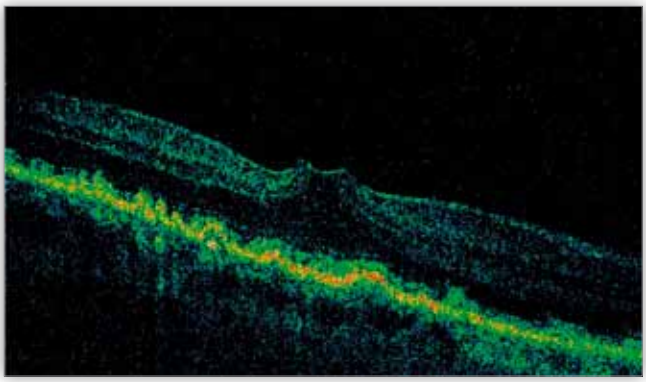
6. Macular hole in OCT

Idiopathic macular hole develops and gradually grows as a result of adjacent retinochoroidal tractions in the macula.

The following stages of the hole can be distinguished:

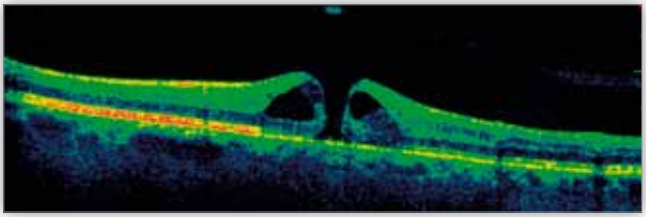
Stage 1a: danger of hole formation, yellow focus in the foveola and disappearance of the physiological depression of the foveola. The nature of this change is rather cyst-like so it is not a real detachment of the sensory retina.

Stage 1b: the hole is hidden in the macula. It results from centrifugal shift of the retinal depression. The characteristic feature of this stage is a yellow ring with a bridge surface of collective interaction with the vitreous body. In 50% of cases it can retreat spontaneously after detaching the vitreous body in the foveola (Fig. 19).



Ryc. 19. Macular hole in ICT, stage 1a: disapperance of the foveal depression with traction of inner limiting membrane of vitreous.
Fig. 19. Otwór plamki w OCT, stadium 1a: zniesienie zagłębienia dołka z trakcją błony granicznej wewnętrznej ciała szklistego.

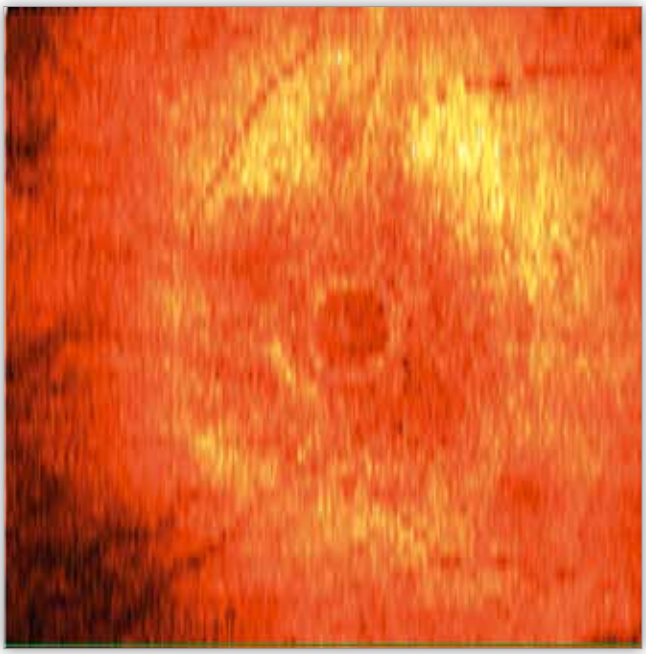
Stage 2: partial-thickness macular hole up to 400 μm in diameter. Pseudooperculum – a concentration in the prefoveola area can be visible (Fig. 20).



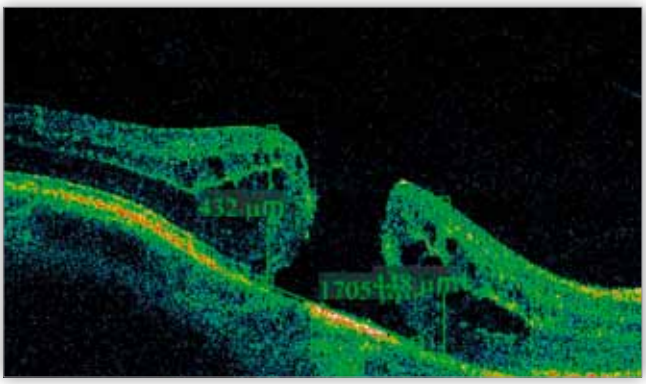
Ryc. 20. Macular hole in OCT, stage 3: hole extending in all retinal layers to retinal pigment with operculum, retinal edema.
Fig. 20. Otwór plamki w OCT, stadium 3.: otwór obejmuje wszystkie warstwy siatkówki do nabłonka barwnikowego, obecne wieczko, obrzęk siatkówki.

Stadium 3: Full-thickness macular hole above 400 μm in diameter with adhering posterior surface of the vitreous. Possible presence of pseudooperculum.

Stage 4: Full-thickness macular hole with a full detachment of the vitreous body (Fig. 21, 22).



Ryc. 21. Color picture of macula – hole in stage 4.
Fig. 21. Kolorowe zdjęcie plamki – otwór w stadium 4.



Ryc. 22. OCT – macular hole in stage 4.
Fig. 22. OCT – otwór plamki w stadium 4.

OCT images show a total lack of reflectivity in the physiological depression. The size of the hole can be measured at its base and from the side of the vitreous body. At the edges of the hole, thickened retina and intraretinal edema are observed. Increased reflectivity of the pigment layer is seen in the location of the hole. The evaluation of hyperreflective, posterior border membrane of the vitreous body and its relation to the retina (detachment or traction) is very important.

At stages 2 and 3 a hyperreflective pseudooperculum can be observed (4.12-14).

7. Diabetic retinopathy in OCT

Diabetic retinopathy is a microangiopathy characterized by occlusion of small vessels and increase in their permeability. Clinically we can divide it into nonproliferative type with limited changes in the retina and proliferative type where the destructive process extends beyond the surface of the retina. Typical changes for the diabetic retinopathy are soft exudates, hard exudates, microaneurysms, intraretinal vascular disorders, hemorrhages and edemas.

Soft exudates are localized superficially and they indicate ischemia in the nerve fiber layer. They are characteristic in preproliferative diabetic retinopathy with extensive areas with no capillary perfusion in fluorescein angiography. In OCT soft exudates take the form of nodular, hyperreflective foci in the nerve fiber layer causing shadows and therefore blocking the reflectivity of the lower retinal layers. Hard exudates are composed of lipoproteins accumulated on the border of ischemic and normal retina. In OCT hard exudates are localized in the inner layers of the retina as small, hyperreflective foci.

Superficial hemorrhages in the course of diabetes are flame-like. They are localized along the nerve fiber, while the hemorrhages in the deeper layers of the capillary tubes are oval or irregular. In OCT the preretinal and subretinal layers are hyperreflective and they cast a shadow over the lower layers.

Macular edema is the main cause of vision impairment at the nonproliferative stage of simple diabetic retinopathy. Usually it appears after 8-10 years of diabetes, however a good control of the metabolism can delay its occurrence. The beginning of the edema is a leak through the walls of the capillary tubes or intraretinal microcapillaries, which are not tight. The edema becomes focal, extended and cyst-like. Focal edema is characterized by local growth of the volume and thickness of the retina with hyporeflective intraretinal spaces in OCT. For diagnostics and monitoring of the edema, maps of the macular retina and fluorescein angiography are very useful. Extended retinal edema is observed in approximately 80% of eyes of diabetic patients and it is usually localized between the external plexal layer and the nucleated internal layer of the retina. The retina in OCT is thickened with low reflectivity. Extended edema takes form of numerous irregular, intraretinal, hyporeflective lacunas. It is accompanied by macular outline distortion and an epiretinal membrane with possible tractions. Long-term extended edema may result in necrosis of Müller cells and formation of cysts in the exterior plexal layer. Pseudocysts are localized in the external layers. Internal layers remain unchanged. When a cyst-like edema lasts for a long time, the walls of pseudocysts break forming big cyst-like cavities filled with fluid. Cyst-like edema results in border atrophy of the retina. It is connected with a major impairment of the vision quality and it can be accompanied by a serous detachment of the retina (Fig. 23-27).

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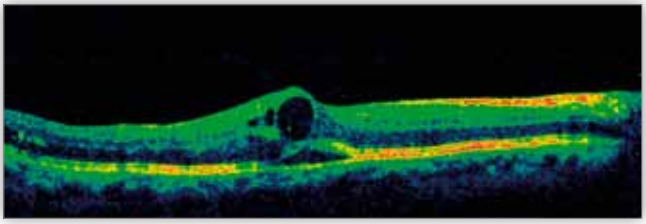
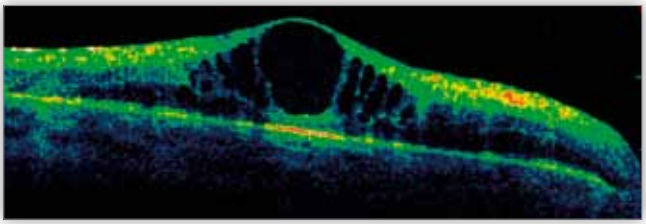
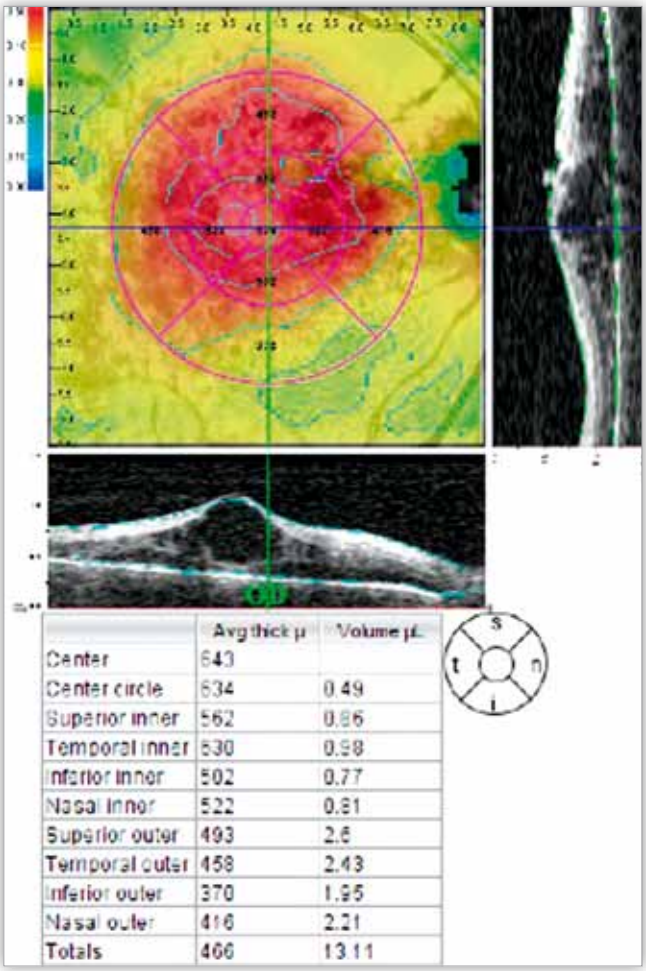


Ryc. 23. Diabetic retinopathy in OCT – cystoid macular edema, subretinal fluid.
Fig. 23. Retinopatia cukrzycowa w OCT – obrzęk cystowaty plamki, płyn pod siatkówką.

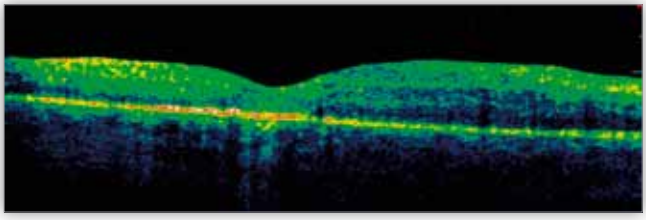


Ryc. 24. Diabetic retinopathy in OCT (B scan) – advance cystoid macular edema before vitreoretinal operation.
Fig. 24. Retinopatia cukrzycowa w OCT (B skan) – zaawansowany obrzęk cystowaty plamki przed operacją witreretinalną.

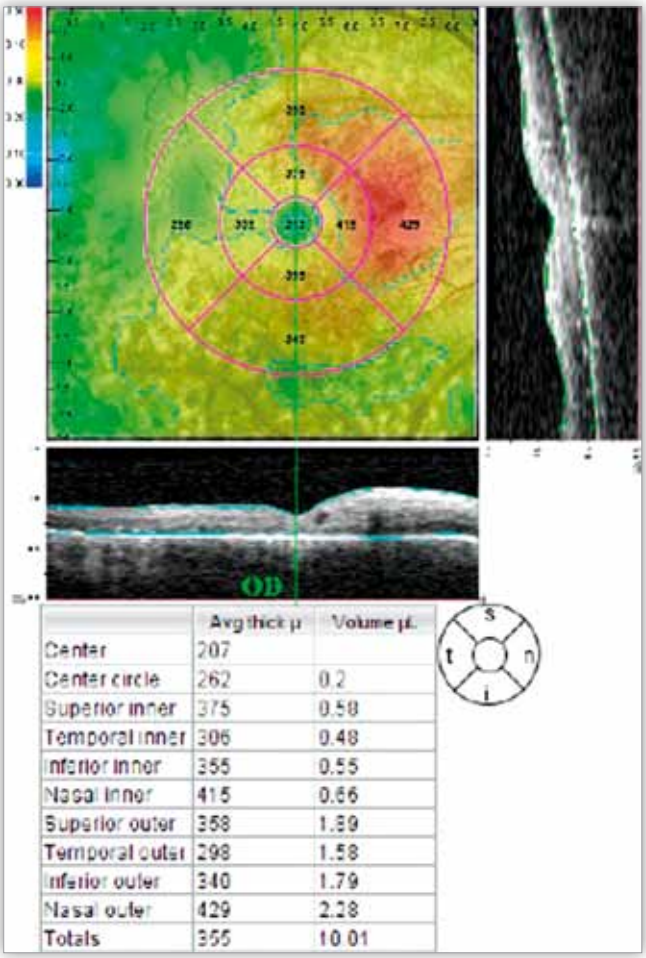


	Avg thick µ	Volume µL
Center	643	
Center circle	634	0.49
Superior inner	562	0.86
Temporal inner	630	0.98
Inferior inner	502	0.77
Nasal inner	522	0.81
Superior outer	493	2.6
Temporal outer	458	2.43
Inferior outer	370	1.95
Nasal outer	416	2.21
Totals	466	13.11

Ryc. 25. Diabetic retinopathy in OCT (3D) – advance cystoid macular edema before vitreoretinal operation.
Fig. 25. Retinopatia cukrzycowa w OCT (3D) – zaawansowany obrzęk cystowaty plamki przed operacją witreretinalną.



Ryc. 26. Scan B after vitreoretinal operation, without retinal edema.
Fig. 26. Skan B po operacji witreoretinalnej, bez obrzęku siatkówki.



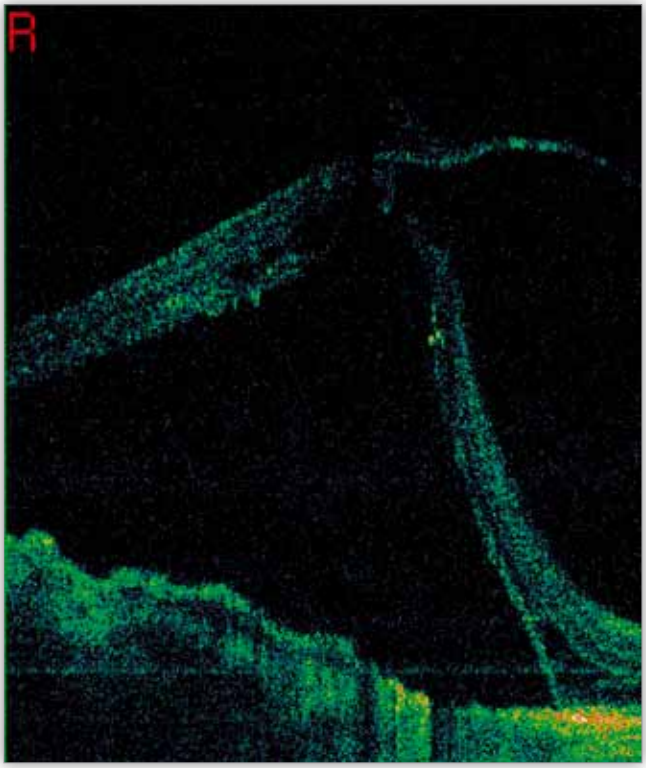
	Avg thick µ	Volume µL
Center	207	
Center circle	262	0.2
Superior inner	375	0.58
Temporal inner	306	0.48
Inferior inner	355	0.55
Nasal inner	415	0.66
Superior outer	358	1.89
Temporal outer	298	1.58
Inferior outer	340	1.79
Nasal outer	429	2.28
Totals	355	10.01

Ryc. 27. 3D picture after vitreoretinal operation.
Fig. 27. Obraz 3D po operacji witreoretinalnej.

In the proliferative stage of diabetic retinopathy, hyperreflective, preretinal or prediscoid neovascular membranes are clearly visible if they contain fibrous components (Fig. 28). They cause retinal tractions and in advanced stages they can result in tractional retinal detachment (4,15-17).

Conclusion

The advantages of optic coherent tomography in the diagnostics of macular diseases are: simplicity of performance and short time of examination, sensitivity, non-invasiveness a lack of contact. There are few obstacles such as corneal edema, cataract, vitreous opacites or hemorrhages. The examination is usually performed after pupillary dilation. The OCT images should be interpreted by an experienced retinologist.



Ryc. 28. Vitreoretinal tractions in OCT.
Fig. 28. Trakcje szklistkowo-siatkówkowe w OCT.

References:

1. Wylęgała E, Teper S, Wróblewska-Czajka E et al.: *Optyczna koherentna tomografia*. Oftal Warszawa 2007, 61-67.
2. Schuman JS, Puliafito CA, Fujimoto JG: *Optical coherence tomography of ocular diseases*. II edition SLACK 2004, 21-57.
3. Gupta V, Gupta A, Dogra MR: *Atlas of optical coherence tomography of macular diseases and glaucoma.* YAYPEE BROTHERS Medical Publishers LTD New Delhi Second Edition 2006, 169-207.
4. Kański JJ, Milewski SA: *Choroby plamki*. I Polish edition edited by Krystyna Pecold. Górnicki Medical Publishin House, Wrocław (Wydawnictwo Medyczne Wrocław) 2003, 19-69.
5. Costa RA, Farah ME, Cardillo JA et al.: *Immediate indocyanine green angiography and optical coherence tomography evaluation after photodynamic therapy for subfoveal choroidal neovascularization*. Retina 2003, 23, 159-165.
6. Puliafito CA, Hee MR, Lin CP et al.: *Imaging of macular disease with optical coherence tomography*. Ophthalmology 1995, 102, 217-219.
7. Massacesi AL, Sacchi L, Bergamini F et al.: *The prevalence of retinal angiomatous proliferation in age-related macular degeneration with occult choroidal neovascularization*. Graefes Arch Clin Exp Ophthalmol 2008, 246, 89-92.
8. Wilkins JR, Puliafito CA, Hee MR et al.: *Characterization of epiretinal membranes using optical coherence tomography*. Ophthalmology 1996, 103, 2142-2151.
9. Hee MR, Puliafito CA, Wong C et al.: *Optical coherence tomography of central serous chorioretinopathy*. Am J Ophthalmol 1995, 120, 65-74.
10. Piccolino FC, de la Longrais RR, Rauera G et al.: *The foveal photoreceptor layer and issued acuity loss in central serous chorioretinopathy*. Am J Ophthalmol 2005, 139, 87-99.
11. Wang M, Larsen M, Sander R et al.: *Central serous chorioretinopathy with foveal detachment demonstrated by optical coherence tomography*. Acta Ophthalmol Scand 1999, 77, 402-405.
12. Chauhan DS, Antcliff RJ, Rai PA et al.: *Papillofoveal traction in macular hole formation*. Arch Ophthalmol 2000, 118, 32-38.
13. Hee MR, Puliafito CA, Carlton W et al.: *Optical coherence tomography of macular holes*. Ophthalmol 1995, 102, 748-756.
14. Ullrich S, Haritoglou C, Gass C et al.: *Macular hole size as a prognostic factor in macular hole surgery*. Br J Ophthalmol 2002, 86, 390-393.
15. Catier A, Tadayoni R, Paques M et al.: *Characterization of macular edema from various etiologies by optic coherence tomography*. Am J Ophthalmol 2005, 140, 200-206.
16. Gaucher D, Tadayoni R, Erginary A et al.: *Optical coherence tomography assessment of the vitreorelationship in diabetic macular edema*. Am J Ophthalmol 2005, 139, 807-813.
17. Goebel W, Kretzchmar-Gross T: *Retinal thickness in diabetic retinopathy: a study using optical coherence tomography*. Retina 2002, 22, 759-767.

The study was originally received 16.06.2009 (1151)/
Praca wpłynęła do Redakcji 16.06.2009 r. (1151)
Accepted for publication 20.03.2010/
Zakwalifikowano do druku 20.03.2010 r.

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ISSN 0023-2157 Index 362646