e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2023; 15(10); 529-534

Original Research Article

Optical Coherence Tomography in Movement Disorders

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Received: 25-07-2023 / Revised: 28-08-2023 / Accepted: 30-09-2023
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Conflict of interest: Nil

Abstract:

The optic nerve head and retina can be visualised in high-resolution two-dimensional cross-sections using optical coherence tomography, a cutting-edge non-invasive imaging technique. The optic nerve head and retina can also be quantified and measured in three dimensions. For the diagnosis and follow-up of several eye illnesses, including glaucoma, diabetic retinopathy, and age-related macular degeneration, ophthalmologists frequently use this technology. Since various clinical studies have shown that these disorders result in reduced thickness of the inner retinal nerve fibre layer, which is mostly made up of retinal ganglion cells and their axons, it has attracted a great deal of attention in recent years. To demonstrate the promise of this non-invasive and widely available technique, we sought to discuss the clinical utility of optical coherence tomography for identifying and assessing various movement disorders in this review.

Keywords: Tomography, Optical Coherence, Huntington's Disease, Parkinson Disease, Wilson's Disease.

Introduction

The substantia nigra pars reticulata (SNpr), one of the basal ganglia's many nuclei, plays a significant role in the development of eye movements. [1] Through its connections to the superior colliculus, the basal ganglia regulate saccadic eye movements (SC). [2] There are two routes in which the frontal eye fields' cortical projections get to the SC. One of these is the excitatory straight route. The SC is reached by the second pathway via a number of synapses in the basal ganglia nuclei. Although the GABAergic output from the basal ganglia ultimately inhibits or disinhibits the neurons in the colliculus, this route is also excitatory. [1] The caudate nucleus transmits inhibitory projections to the substantia nigra, which then exerts more inhibition on the SC, causing the SC to briefly become disinhibited and for the brainstem nuclei to produce a saccade. [3-5]

The eye can be affected by both hypokinetic and hyperkinetic movement disorders. [6,7] Disfunction at the level of the retina, optic nerves, oculomotor system, or subcortical or visual cortex can lead to ophthalmological symptoms. [8] Ophthalmological data provide insight into the severity of distinct movement disorders and aid in their differentiation. These come after or could come before a movement disorder diagnosis. With particular reference to Parkinson's disease (PD) and Huntington's disease, eye movement abnormalities using quantitative recording techniques are now being considered as non-invasive biomarkers to study the natural progression of disease and effects of various therapies in various movement disorders. [9-13] Instead, abnormal eye movements have diagnostic and prognostic implications and have the potential to further research.

The optic nerve head and retina can be imaged with high-resolution, two-dimensional cross-sectional pictures and quantitative, reproducible three-dimensional volumetric data using optical coherence tomography (OCT), a contemporary non-invasive imaging technique [14]. For the diagnosis and follow-up of several eye illnesses, including glaucoma, diabetic retinopathy, and age-related macular degeneration, ophthalmologists frequently use this technology. Since various clinical investigations have shown that neurodegenerative disorders result in decreased thickness of the inner retinal nerve fibre layer (RNFL), which is mostly made up of retinal ganglion cells and their axons, these diseases have attracted a great deal of attention in recent years [15]. Changes in retinal vascular density may also have potential as an ocular biomarker for neurodegenerative diseases due to the similarities between the microvascular structures of the retina and the brain and the presence of vascular abnormalities in the brain in many patients with neurodegenerative disorders. The OCT-angiography (OCTA) approach uses light laser

reflectance to detect the movement of intravascular red blood cells and rebuild the retinal microvasculature in great detail without the use of contrast. It is a non-invasive method for imaging the retina's microvasculature. [16] We will talk about OCT findings in movement disorders in this review.

Principles of Optical Coherence Tomography (OCT)

OCT is a noninvasive diagnostic technique that generates in vivo cross-sectional images of the retina. It uses near-infrared light, based on low-coherence interferometry, to create a cross-sectional image of the retina. The first commercially available versions of OCT used time-domain (TD-OCT) technology, which requires long acquisition times and provides axial and lateral resolutions of 15 mm. Improvements have been achieved over the past two decades with the emergence of spectral-domain OCT (SD-OCT) technology. This provides three-dimensional high-resolution cross-sectional retinal images with an axial resolution up to five times greater and imaging speeds approximately 60 times greater than TD-OCT.[17]

The retinal layers are automatically identified by the OCT device. It considers the differences in reflectivity and signals generated by each retinal layer. Thus, peripapillary RNFL (pRNFL) is defined as the distance between the internal limiting membrane and the retinal ganglion cell/inner plexiform layer. These layers are located between the RNFL and the inner nuclear layer. The total macular thickness is calculated considering the distance between the internal limiting membrane and the retinal pigment epithelium. [17]

The pRNFL and macula thickness measures are both automatically estimated by the OCT equipment. The majority of OCT equipment acquire pRNFL thickness measurement reports by scanning a 6 6 mm area with 521 A-scans horizontally and 256 A-scans vertically, with the optic nerve head in the centre. A circle with a diameter of 3.4 mm that is centred on the optic nerve head makes up the measurement area. The thicknesses of the 12 clock hour segments, the four quadrants (superior, temporal, inferior, and nasal), and the average thickness are all measured (in mm). The macular analysis techniques include 512 A-scans horizontally and 128 A-scans vertically in a scanned area measuring 6 mm by 6 mm. Based on a 6 mm circular map segmented into nine segments, the macular analysis is performed. The average macular thickness and the thicknesses of each of the nine sectors were measured (in mm) [17].

Optical Coherence Tomography in Parkinson's Disease

The most prevalent form of parkinsonism is PD, which is also the second most prevalent neurodegenerative illness affecting the elderly. Visual dysfunction (visual acuity, dynamic contrast sensitivity, and colour discrimination), pupil abnormality, lens opacity, motion perception, visual processing speeds, facial recognition issues, chronic visual hallucinations, and loss and dysfunction of retinal neuronal cells are just a few of the ocular changes that have been linked to PD [18,19]. The distinguishing clinical characteristics of Parkinson's disease (PD) in the brain are assumed to be mirrored by these visual abnormalities, which are connected to -synuclein accumulation and dopamine insufficiency in the retina [18,19].

An abundant neuronal protein called alpha-synuclein controls the movement of synaptic vesicles and the consequent release of neurotransmitters. Under pathological circumstances, it aggregates to form insoluble fibrils called synucleinopathies, which can cause a variety of cellular problems. A distinct group of amacrine cells in the inner nuclear layer release dopamine, which activates D1 and D2 dopamine receptors found all over the retina. Most frequently, decreased colour vision, visual contrast sensitivity, and visual acuity have been linked to decreased retinal dopamine [20]. Visual impairment has generally been thought to be a result of PD development, although there are some visual characteristics that can be seen in early PD and may even be present in the prodromal phase.

Recent developments have increased interest in the role of the retina as a potential biomarker for making an early diagnosis of PD, as well as a way to track the development of the condition and assess cutting-edge treatment options. OCT may be used as a potential biomarker for early PD diagnosis and prognosis if early dopamine failure results in structural abnormalities of the retina that may be seen through retinal imaging.

Inzelberg et al. [21] were the first to demonstrate RNFL thinning in ten PD patients using OCT in 2004. Their findings were supported by further research, which also revealed decreases in macular volume and thickness [22,23]. Nonetheless, according to a number of additional investigations [24–26], RNFL thickness was comparable between patients and controls. The RNFL thickness was found to have a negative correlation with the severity of PD as determined by the Hoehn-Yahr stage and the Unified Parkinson's Disease Rating Scale (UPDRS) scores [24,25,27]. The akinetic-rigid subtype of PD was found to have thinner RNFL than the tremor-predominant subtype [28]. Moreover, cognition [29] and the prevalence of hallucinations [26] have been linked to RNFL thickness. A progressive decline in RNFL thickness

was observed in recent longitudinal investigations, and this was accompanied by a progressive decline in visual function [30,31].

Better understanding of retinal thinning in PD might be obtained by segmented retinal layer measurements. According to a recent meta-analysis, the macular layers most commonly impacted by PD are the inner plexiform layer and the ganglion cell layer [32]. Retinal changes in PD patients point to vascular and dopaminergic pathways, but further research is required to substantiate this notion. OCT assessments in PD patients present a number of problems, although pooled data from a recent systematic analysis indicated that there are strong relationships between retinal OCT measurements and PD [33], highlighting the potential value of OCT as an imaging biomarker in PD. Moreover, combining OCT and OCTA may improve diagnostic performance compared to any test used alone, offering new biomarker techniques for tracking the start and progression of Parkinson's disease [34]. In comparison to age-matched healthy controls, PD patients' PRNFL thickness was significantly reduced according to OCT investigations. All of the industries examined experienced a sizable thinning. Unlike to other neurodegenerative disorders like Alzheimer's disease, the thinning was not uniform and was more pronounced on the temporal hemiretina than the nasal half. Ten PD patients were shown to have RNFL thinning by OCT by Inzelberg et al. in 2004. [21] Thereafter, numerous more groups, including Yavas et al. in 2007, showed comparable outcomes. [35] In their study of 17 PD and 11 controls the following year, Altintas et al. reported reduced mean RNFL thicknesses in all quadrants, with the exception of the 8 o'clock position, when compared to control subjects. [22] In a 2012 study of 42 PD patients, Kirbas et al. discovered a comparable differential hemiretinal thinning of the temporal retina. [36] A 2011 study by Moschos et al. on 16 PD patients produced corroborating results. However, they also discovered differential thinning in the inferior quadrant along with temporal hemiretina. [37] Yet, neither of them was able to provide a detailed justification for the same.

OCT in Progressive Supranuclear Palsy

A tauopathy called progressive supranuclear palsy (PSP) is frequently characterised by frontal lobe involvement, symmetrical parkinsonism, axial rigidity, falls in the first year of the disease, and retrocollis. [38] Due to the site of its pathology, it is well-known for a wide range of ocular abnormalities. The slowing of vertical saccades is the first anomaly of eye movements identified in PSP. The disease's distinguishing feature is believed to be vertical supranuclear gaze palsy. [39] Optokinetic nystagmus is best for detecting abnormalities in downhill saccades. [40] However, in the latter phases, which eventually result in total ophthalmoplegia, horizontal saccades are also compromised.

Spectral domain optical coherence tomography has also been used in PSP to identify changes in retinal morphology (SD-OCT). [41] In 22 PSP patients, Stemplewitz et al.[42] found reduced macular thickness as well as inferior nasal and inferior temporal retinal thinning.

The severity or duration of the condition did not, however, correspond with retinal alterations. In order to distinguish PSP from PD, Albrecht et al. showed that the ratio of the outer nuclear layer to the outer plexiform layer, with a cutoff of 3.1 and a thickness of the inner nuclear layer less than 46 m, had a sensitivity of 96% and a specificity of 70%.

OCT in Corticobasal Syndrome

Cortical myoclonus, alien limb experience, cortical sensory loss, and gradually progressing asymmetrical cortical and extrapyramidal dysfunction are all symptoms of corticobasal syndrome. [43]

It is a pathologically varied entity that might appear as PSP, Corticobasal Ganglionic Degeneration, Picks Disease, Alzheimer's Disease, Frontotemporal Lobar Degeneration, Dementia with Lewy Bodies, or Creutzfeldt-Jakob Disease. [44] Using spectral domain OCT, Albrecht et al. [24] examined the thickness of the retinal nerve fibre layer in 10 patients with corticobasal syndrome. The scientists discovered significant variations in macular thickness and retinal characteristics in these patients, which may be related to several underlying diseases. Increased outer nuclear layer thickness was a remarkable discovery.

OCT in Huntington’s Disease

The Huntingtin gene on chromosome 4 has an enlarged cytosine-adenine-guanine (CAG) repeat, which results in Huntington's disease, an autosomal dominant illness. It is characterised by a triad of movement problems, cognitive decline, and neuropsychiatric indications and is the most frequent cause of genetic chorea. [45] Using spectral domain optical coherence tomography, Kersten et al.[46] examined the morphology of the optic nerve and macula in 39 patients with Huntington's disease.

The temporal sector and macula both have thinner retinal nerve fibre layers, according to the authors. Macular volumes and temporal retinal nerve fibre layer thickness were shown to be significantly inversely correlated with illness duration and severity.

OCT in Spinocerebellar Ataxias

Spinocerebellar ataxias, which demonstrate clinical, pathological, and genetic variety but are marked by numerous extrapyramidal symptoms in addition to ataxia, are autosomal dominant trinucleotide repeat disorders. [47] OCT was done on 24 patients with genetically confirmed SCA by Pula et al.[78]. In comparison to controls, the authors discovered considerable thinning at the peripapillary retinal nerve fibre layer in SCA2 and SCA3, and perifoveal macula thinning in SCA1, SCA3, and SCA6. [48] In patients with SCA 2 and SCA 3, a strong inverse relationship between illness severity as determined by the SARA score (Scale for the Assessment and Rating of Ataxia) and retinal nerve fibre layer thickness was discovered. [48]

OCT in Wilson's Disease

Wilson's illness is a condition of copper metabolism that has extensive, primarily hepatic and neurological effects on the body. Tremor, dystonia, ataxia, and parkinsonism are the characteristics of the neurological symptoms. [49] Wilson's illness can cause visual impairment as a result of retinal degeneration and vitamin A malabsorption. Electrophysiological experiments using pattern reversal visual evoked potential recordings and electroretinograms have shown extended latencies with reduced amplitudes. With decoppering therapy, these anomalies also improve in part parallel to the clinical improvement. [50]

Wilson's disease patients' retinal degeneration has been seen in optical coherence tomography studies as reduced thickness in the peripapillary retinal nerve fibre layer, macula, inner plexiform layer, and inner nuclear layer. [51] Another study of 58 individuals discovered a substantial negative connection between OCT parameters and neurological severity in addition to showing reduced thickness of the retinal nerve fibre layer and macula. [52] Moreover, patients with positive MRI results have significantly lower retinal nerve fibre layers and macular volumes than patients with negative MRI results, according to Langwinska et al. [53].

Conclusion

In conclusion, optical coherence tomography (OCT) is a useful noninvasive technology for identifying and monitoring neuroaxonal loss in various neurodegenerative illnesses and may be utilised to offer these patients with an easily accessible ocular biomarker. Movement problems can impact the optic nerves, retina, visual brain, and, most significantly, the ocular motor system. An eye exam is crucial in the realm of movement disorders. It aids in reducing the range of possible diagnoses in difficult cases with both hypokinetic and hyperkinetic movement disorders. The function of the retina in neurodegenerative diseases and its significance as a biomarker are now being clarified by recent studies. So, when assessing movement problems, it is essential to be familiar with the many facets of the neuroophthalmological examination.

References

1. Basso MA, Sommer MA. Exploring the role of the substantia nigra pars reticulata in eye movements. Neuroscience 2011;198:205-12.
2. Shires J, Joshi S, Basso MA. Shedding new light on the role of the BG-SC pathway in eye movements. Curr Opin Neurobiol 2010; 20:717-25.
3. Hikosaka O, Takikawa Y, Kawagoe R. Role of the basal ganglia in the control of purposive saccadic eye movements. Physiol Rev 2000;80:953-78.
4. Puri S, Shaikh AG. Basic and translational neuro-ophthalmology of visually guided saccades: Disorders of velocity. Expert Rev Ophthalmol 2017;12:457-73.
5. Watanabe M, Munoz DP. Probing basal ganglia functions by saccade eye movements. Eur J Neurosci 2011;33:2070-90.
6. Lal V, Truong D. Eye movement abnormalities in movement disorders. Clin Park Relat Disord 2019;1:54-63.
7. Jung I, Kim J-S. Abnormal eye movements in parkinsonism and movement disorders. J Mov Disord 2019;12:1-13.
8. Termsarasab P, Thammongkolchai T, Rucker JC, Frucht SJ. The diagnostic value of saccades in movement disorder patients: A practical guide and review. J Clin Mov Disord. 2015;2:1-10.
9. Shaikh AG, Zee DS. Eye movement research in the twenty-first century—a window to the brain, mind, and more. Cerebellum 2018;17:252-8.
10. Blekher T, Weaver M, Rupp J, Nichols WC, Hui SL, Gray J, et al. Multiple step pattern as a biomarker in Parkinson disease. Park Relat Disord 2009;15:506-10.
11. Michell AW, Xu Z, Fritz D, Lewis SJG, Foltynie T, WilliamsGray CH, et al. Saccadic latency distributions in Parkinson's disease and the effects of L-DOPA. Exp Brain Res 2006;174:7-18.
12. Antoniades CA, Xu Z, Mason SL, Carpenter RHS, Barker RA. Huntington's disease: Changes in saccades and hand-tapping over 3 years. J Neurol 2010;257:1890-8.
13. Anderson TJ, MacAskill MR. Eye movements in patients with neurodegenerative disorders. Nat Rev Neurol 2013;9:74-85.
14. Huang D, Swanson EA, Lin CP, Schuman JS, Stinson WG, Chang W, et al. Optical coherence tomography. Science. 1991 Nov; 254(5035): 1178-81.

15. Chan VTT, Sun Z, Tang S, Chen LJ, Wong A, Tham CC, et al. Spectraldomain OCT measurements in Alzheimer's disease: a systematic review and meta-analysis. Ophthalmology. 2019 Apr;126(4):497-510.
16. Fingler J, Readhead C, Schwartz DM, Fraser SE. Phase-contrast OCT imaging of transverse flows in the mouse retina and choroid. Invest Ophthalmol Vis Sci. 2008 Nov;49(11):5055-9.
17. Costa-Cunha LV, Cunha LP, Malta RF, Monteiro ML. Comparison of Fourier-domain and time-domain optical coherence tomography in the detection of band atrophy of the optic nerve. Am J Ophthalmol 2009 Jan;147(1):56-63.
18. Armstrong RA. Visual signs and symptoms of Parkinson's disease. Clin Exp Optom. 2008 Mar; 91(2):129-38.
19. Weil RS, Schrag AE, Warren JD, Crutch SJ, Lees AJ, Morris HR. Visual dysfunction in Parkinson's disease. Brain. 2016 Nov; 139(11): 2827- 43.
20. Price MJ, Feldman RG, Adelberg D, Kayne H. Abnormalities in color vision and contrast sensitivity in Parkinson's disease. Neurology. 1992 Apr;42(4):887-90.
21. Inzelberg R, Ramirez JA, Nisipeanu P, Ophir A. Retinal nerve fiber layer thinning in Parkinson disease. Vision Res. 2004 Nov; 44(24):2793-7.
22. Altintaş O, Işeri P, Ozkan B, Cağlar Y. Correlation between retinal morphological and functional findings and clinical severity in Parkinson's disease. Doc Ophthalmol. 2008 Mar;116(2):137-46.
23. Hajee ME, March WF, Lazzaro DR, Wolintz AH, Shrier EM, Glazman S, et al. Inner retinal layer thinning in Parkinson disease. Arch Ophthalmol. 2009 Jun;127(6):737-41.
24. Albrecht P, Muller AK, Sudmeyer M, Ferrea S, Ringelstein M, Cohn E, et al. Optical coherence tomography in parkinsonian syndromes. PLoS One. 2012 Apr;7(4):e34891.
25. Mailankody P, Battu R, Khanna A, Lenka A, Yadav R, Pal PK. Optical coherence tomography as a tool to evaluate retinal changes in Parkinson's disease. Parkinsonism Relat Disord. 2015 Oct;21(10):1164-9.
26. Lee JY, Kim JM, Ahn J, Kim HJ, Jeon BS, Kim TW. Retinal nerve fiber layer thickness and visual hallucinations in Parkinson's Disease. Mov Disord. 2014 Jan;29(1):61-7.
27. Rohani M, Langroodi AS, Ghourchian S, Falavarjani KG, SoUdi R, Shahidi G. Retinal nerve changes in patients with tremor dominant and akinetic rigid Parkinson's disease. Neurol Sci. 2013 May;34(5):689-93.
28. Moschos MM, Chatziralli IP. Evaluation of choroidal and retinal thickness changes in parkinson's disease using spectral domain optical coherence tomography. Semin Ophthalmol. 2018;33(4):494-7.
29. Yildiz D, Pekel NB, Yener NP, Seferoglu M, Gunes A, Sigirli D. Assessment of neurodegeneration by optical coherence tomography and mini-mental test in Parkinson's disease. Ann Indian Acad Neurol. 2019 Apr-Jun;22(2):212-6.
30. Ma LJ, Xu LL, Mao CJ, Fu YT, Ji XY, Shen Y, et al. Progressive changes in the retinal structure of patients with Parkinson's disease. J Parkinsons Dis. 2018;8(1):85-92.
31. Satue M, Rodrigo MJ, Obis J, Vilades E, Gracia H, Otin S, et al. Evaluation of progressive visual dysfunction and retinal degeneration in patients with Parkinson's disease. Invest Ophthalmol Vis Sci. 2017 Feb;58(2):1151-7.
32. Chrysou A, Jansonius NM, van Laar T. Retinal layers in Parkinson's disease: A meta-analysis of spectral-domain optical coherence tomography studies. Parkinsonism Relat Disord. 2019 Jul;64:40-49.
33. Zhou WC, Tao JX, Li J. Optical coherence tomography measurements as potential imaging biomarkers for Parkinson's disease: A systematic review and meta-analysis. Eur J Neurol. 2020 Mar;28(3):763-74.
34. Zou J, Liu K, Li F, Xu Y, Shen L, Xu H. Combination of optical coherence tomography (OCT) and OCT angiography increases diagnostic efficacy of Parkinson's disease. Quant Imaging Med Surg. 2020 Oct;10(10):1930-9.
35. Yavas GF, Yilmaz O, Küsbeci T, Oztürk F. The effect of levodopa and dopamine agonists on optic nerve head in Parkinson disease. Eur J Ophthalmol 2007;17:812-6.
36. Kirbas S, Turkyilmaz K, Tufekci A, Durmus M. Retinal nerve fiber layer thickness in Parkinson disease. J Neuroophthalmol 2013;33:62-5.
37. Moschos MM, Tagaris G, Markopoulos I, Margetis I, Tsapakis S, Kanakis M, et al. Morphologic changes and functional retinal impairment in patients with Parkinson disease without visual loss. Eur J Ophthalmol 2011;21:24-9.
38. Litvan I, Agid Y, Calne D, Campbell G, Dubois B, Duvoisin RC, et al. Clinical research criteria for the diagnosis of progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome): Report of the NINDS-SPSP international workshop. Neurology 1996;47:1-9.
39. Phokaewvarangkul O, Bhidayasiri R. How to spot ocular abnormalities in progressive supranuclear palsy? A practical review. Transl Neurodegener 2019;8:1-14.
40. Garbutt S, Riley DE, Kumar AN, Han Y, Norwood MB, Leigh RJ. Abnormalities of

optokinetic nystagmus in progressive supranuclear palsy. J Neurol Neurosurg Psychiatry 2004;75:1386- 94.
41. Gulmez Sevim D, Unlu M, Gultekin M, Karaca C, Mirza M, Mirza GE. Evaluation of retinal changes in progressive supranuclear palsy and Parkinson disease. J Neuroophthalmol 2018;38:151-5.
42. Stemplewitz B, Kromer R, Vettorazzi E, Hidding U, Frings A, Buhmann C. Retinal degeneration in progressive supranuclear palsy measured by optical coherence tomography and scanning laser polarimetry. Sci Rep 2017;7:1-10.
43. Whitwell JL, Jack CR, Boeve BF, Parisi JE, Ahlskog JE, Drubach DA, et al. Imaging correlates of pathology in corticobasal syndrome. Neurology 2010;75:1879-87.
44. Boeve BF. The multiple phenotypes of corticobasal syndrome and corticobasal degeneration: Implications for further study. J Mol Neurosci 2011;45:350-3.
45. Videnovic A, Shannon KM. Huntington disease and other choreas. Hyperkinetic Mov Disord 2012;23-54.
46. Kersten HM, Danesh-Meyer HV, Kilfoyle DH, Roxburgh RH. Optical coherence tomography findings in Huntington's disease: A potential biomarker of disease progression. J Neurol 2015;262:2457-65.
47. Soong BW, Morrison PJ. Spinocerebellar ataxias. 1st ed. Vol. 155. Handbook of Clinical Neurology. Elsevier B.V.; 2018. pp. 143-74.
48. Pula JH, Towle VL, Staszak VM, Cao D, Bernard JT, Gomez CM. Retinal nerve fibre layer and macular thinning in spinocerebellar ataxia and cerebellar multisystem atrophy. Neuro-Ophthalmology 2011;35:108-14.
49. Aggarwal A, Bhatt M. The pragmatic treatment of Wilson's disease. Mov Disord Clin Pract 2014;1:14-23.
50. Satishohandra P, Ravishankar Naik K. Visual pathway abnormalities Wilson's disease: an electrophysiological study using electroretinography and visual evoked potentials. J Neurol Sci 2000;176:13-20.
51. Albrecht P, Müller AK, Ringelstein M, Finis D, Geerling G, Cohn E, et al. Retinal neurodegeneration in Wilson's disease revealed by spectral domain optical coherence tomography. PLoS One 2012;7:14-6.
52. Langwińska-Wośko E, Litwin T, Dzieżyc K, Karlinski M, Członkowska A. Optical coherence tomography as a marker of neurodegeneration in patients with Wilson's disease. Acta Neurol Belg 2017;117:867-71.
53. Langwińska-Wośko E, Litwin T, Szulborski K, Członkowska A. Optical coherence tomography and electrophysiology of retinal and visual pathways in Wilson's disease. Metab Brain Dis 2016;31:405-15.