DOI: 10.14744/eer.2024.80775
Eur Eye Res 2024;4(0):00–00

EUROPEAN EYE RESEARCH

ORIGINAL ARTICLE

Selective serotonin reuptake inhibitors and ocular health: Analyzing retinal and choroidal thickness variations

Ibrahim Edhem Yilmaz,[1] Kadir Erdogan Er,[3] Necip Kara[2]

[1]Department of Ophthalmology, Gaziantep Islam Science and Technology University, Gaziantep, Türkiye
[2]Department of Ophthalmology, Gaziantep University, Gaziantep, Türkiye
[3]Veni Vidi Eye Hospital, İstanbul, Türkiye

Abstract

Purpose: This study evaluated the posterior segment parameters of the eye in patients using selective serotonin reuptake inhibitors (SSRIs) without systemic disease, using spectral-domain optical coherence tomography (SD-OCT), and compared the effects of different durations of SSRI use on the eye.

Methods: The study involved 104 participants, divided into three groups: those using SSRIs for less than a year (group 1a), those using SSRIs for 1 year or longer (group 1b), and healthy controls (group 2). The posterior segment parameters of the eye were measured using the SD-OCT, and the data were analyzed using descriptive statistics, analysis of variance, post hoc tests, and correlation analysis.

Results: The results showed that the retinal nerve fiber layer (RNFL) thickness and central foveal thickness (CFT) were significantly lower in Group 1a than in Group 2 ($p<0.05$), while there was no significant difference between Group 1b and Group 2 ($p>0.05$). The choroidal thickness was significantly lower in both Group 1a and Group 1b than in Group 2 ($p<0.05$), but there was no significant difference between the two patient groups ($p>0.05$). The axial length (AXL) was not significantly different among the groups ($p>0.05$). There was a weak negative correlation between the duration of SSRI use and the RNFL thickness ($r=-0.25$, $p=0.039$), and a moderate negative correlation between the duration of SSRI use and the CFT ($r=-0.37$, $p=0.002$). There was no significant correlation between the duration of SSRI use and the choroidal thickness or the AXL ($p>0.05$).

Conclusion: This study suggests that SSRIs may affect the retina and choroid due to various mechanisms. The effects may be time dependent and dose dependent, with longer-term use potentially causing adaptations. Ophthalmologists and psychiatrists should monitor patients for symptoms.

Keywords: Antidepressant, choroid, retina, retinal nerve fiber layer, selective serotonin reuptake inhibitor

Selective serotonin reuptake inhibitors (SSRIs) are a mainstay in depression treatment. Their mechanism of action involves blocking serotonin reuptake, leading to increased levels of this neurotransmitter in the synaptic space. This alteration in serotonin activity is believed to be responsible for both the therapeutic effects on depression and the occurrence of various side effects. Notably, the frequency of these side effects appears to be dose-dependent, with a higher incidence at increased dosages. In addition, the widespread use of SSRIs has led to a rise in the number of rare side effects being reported.[1-3]

The well-documented side effects of SSRIs include

Cite this article as: Yilmaz IE, Er KE, Kara N. Selective serotonin reuptake inhibitors and ocular health: Analyzing retinal and choroidal thickness variations. Eur Eye Res 2024;4(0):00–00.

Correspondence: Ibrahim Edhem Yilmaz, M.D. Department of Ophthalmology, Gaziantep Islam Science and Technology University, Gaziantep, Türkiye
E-mail: ibrahimedhem.yilmaz@gibtu.edu.tr

Submitted Date: 17.04.2024 **Revised Date:** 10.05.2024 **Accepted Date:** 17.06.2024 **Available Online Date:** 00.00.2024

headaches, dizziness, gastrointestinal and central nervous system disturbances, endocrine and allergic issues, serotonin syndrome, and withdrawal symptoms.[1-5] However, recent research has shifted focus to the potential impact of SSRIs on ocular health. Studies suggest that SSRI use might be linked to eye-related side effects such as glaucoma, dry eyes, optic nerve damage, macular degeneration, and choroidal abnormalities.[6-9] It is important to note that these findings are primarily based on case reports and studies with small patient groups, warranting further investigation with larger cohorts. This study aims to address this knowledge gap by analyzing the posterior segment parameters of the eye in patients taking SSRIs for depression, excluding those with any other systemic conditions.

Materials and Methods

Study Design and Groups

This cross-sectional investigation was conducted at a university hospital in southern Turkey between February and September 2018. The study was approved by the university's Ethics Committee (2017/41) and written consent was obtained from all patients and volunteers in accordance with the Declaration of Helsinki of the World Medical Association. Group 1 of the research consisted of depressed patients who received SSRI drug therapy as an antidepressant. In this study, individuals who used medications other than SSRIs, had non-psychiatric diseases, were under 18 years old, underwent eye surgeries (such as phacoemulsification, trabeculectomy, or vitrectomy), or had severe eye conditions (such as glaucoma, uveitis, SMD, or choroidopathy) were excluded from the study. The study cohort was divided into two subgroups based on drug use duration: Group 1a consists of patients who have used the drug for <1 year, while Group 1b consists of patients who have used it for 1 year or longer. The control group (Group 2) consisted of volunteers over the age of 18 who lacked systemic or ocular disease and who did not use medications.

Ocular Examination

All individuals had their refractions checked before and after cycloplegia, and their visual acuities were measured. A detailed biomicroscopic assessment of the anterior and posterior segments was carried out. Goldmann applanation tonometry was used to measure intraocular pressure. The retinal nerve fiber layer (RNFL) thickness was calculated automatically using the spectral domain optical coherence tomography (OCT) instrument (Heidelberg Engineering, Heidelberg, Germany) in five sectors: Nasal, superior, temporal, inferior, and average. The apparatus measured the central foveal thickness (CFT) automatically along the line centered on the fovea. The device was placed in enhanced depth imaging (EDI) mode and measurements were taken from three separate points: The fovea center, 1000 microns temporal of the fovea center, and 1000 microns nasal to the fovea center. To eliminate diurnal change in choroidal thickness, OCT imaging was carried out between 11 a.m. and 13 p.m. In addition, patients were subjected to a thorough ophthalmological examination using the Nidek US-500 (Gamagori, Japan) apparatus, which included axial lengths (AXL). The same ophthalmologist performed all of the tests and measures without knowing which group the patients were in.

Statistical Analysis

The data were analyzed using the Statistical Package for the Social Sciences version 25.0 (IBM Corp., Armonk, NY, USA). The descriptive statistics of the data were calculated using average, standard deviation, and percentage values. The Kolmogorov–Smirnov test was utilized to assess the data's suitability for normal distribution. We used parametric analyses because the distribution was normal. To compare categorical variables, the Chi-square test was used. Using the t-test for independent samples, parameters between groups were compared. Using Pearson's correlation test, the relationship between the data was evaluated. The level of significance was set at $p<0.05$.

Results

The descriptive statistics of the study variables are shown in Table 1. The mean age of the participants was 38.7±11.1 years, and 53.8% of them were female. There was no significant difference in age or gender distribution among the groups ($p>0.05$). The mean duration of SSRI use in Group 1 was 18.6±9.5 months, and there was a significant difference between Group 1a and Group 1b in terms of drug use duration ($p<0.001$).

The results of the analysis of variance and post hoc tests for the comparison of the groups in terms of RNFL thickness, CFT, choroidal thickness, and AXL are shown in Table 2. The RNFL thickness and CFT were significantly lower in Group 1a than in Group 2 ($p<0.05$), while there was no significant difference between Group 1b and Group 2 ($p>0.05$). The choroidal thickness was significantly lower in both Group 1a and Group 1b than in Group 2 ($p<0.05$), but there was no significant difference between the two patient groups

Table 1. Descriptive statistics of the study variables

Variable	Group 1a (n=26)	Group 1b (n=26)	Group 2 (n=52)	p
Age (years)	39.3±10.7	38.1±11.6	38.4±11.2	0.861
Gender (female/male)	14/12	15/11	28/24	0.924
Duration of SSRI use (months)	9.4±2.6	27.8±6.3	-	<0.001
RNFL thickness (µm)	91.6±8.8	95.9±9.1	99.7±7.5	0.002
CFT (µm)	227.2±18.3	235.1±19.4	244.1±16.2	0.001
Choroidal thickness (µm)	251.8±35.4	256.7±37.6	288.9±32.1	<0.001
AXL (mm)	23.4±0.9	23.6±0.8	23.7±0.7	0.541

SSRI: Selective serotonin reuptake inhibitor, RNFL: Retinal nerve fiber layer, CFT: Central foveal thickness, AXL: Axial lenght, µm: Micrometer.

Table 2. Analysis of variance and post hoc tests for the comparison of the groups

Variable	F	p	Post hoc tests
RNFL thickness	6.21	0.002	Group 1a<Group 2* Group 1b=Group 2 Group 1a=Group 1b
CFT	7.01	0.001	Group 1a<Group 2* Group 1b=Group 2 Group 1a=Group 1b
Choroidal thickness	13.87	<0.001	Group 1a<Group 2* Group 1b<Group 2* Group 1a=Group 1b
AXL	0.32	0.541	Group 1a=Group 2 Group 1b=Group 2 Group 1a=Group 1b

SSRI: Selective serotonin reuptake inhibitor, RNFL: Retinal nerve fiber layer, CFT: Central foveal thickness, AXL: Axial length. *=P<0.05.

(p>0.05). The AXL was not significantly different among the groups (p>0.05).

The results of the correlation analysis between the duration of SSRI use and the posterior segment parameters are shown in Table 3. There was a weak negative correlation between the duration of SSRI use and the RNFL thickness (r=–0.25, p=0.039), and a moderate negative correlation between the duration of SSRI use and the CFT (r=–0.37, p=0.002). There was no significant correlation between the duration of SSRI use and the choroidal thickness or the AXL (p>0.05).

The comparison of the RNFL thickness in different sectors among the groups is shown in Table 4. The RNFL thickness in the nasal, superior, temporal, and inferior sectors was significantly lower in Group 1a than in Group 2 (p<0.05), while there was no significant difference between Group 1b and Group 2 (p>0.05). There was no significant difference between the two patient groups in any of the sectors (p>0.05).

Table 3. Correlation analysis between the duration of SSRI use and the posterior segment parameters

Variable	r	p
RNFL thickness	–0.25	0.039
CFT	–0.37	0.002
Choroidal thickness	–0.13	0.256
AXL	–0.09	0.457

SSRI: Selective serotonin reuptake inhibitor. RNFL: Retinal nerve fiber layer. CFT: Central foveal thickness. AXL: Axial length.

The comparison of the CFT in different points among the groups is shown in Table 5. The CFT at the fovea center, 1000 µm temporal, and 1000 µm nasal were significantly lower in Group 1a than in Group 2 (p<0.05), while there was no significant difference between Group 1b and Group 2 (p>0.05). There was no significant difference between the two patient groups at any of the points (p>0.05).

The comparison of the choroidal thickness in different points among the groups is shown in Table 6. The choroidal thickness at the fovea center, 1000 µm temporal, and 1000 µm nasal were significantly lower in both Group 1a and Group 1b than in Group 2 (p<0.05), but there was no significant difference between the two patient groups at any of the points (p>0.05).

Discussion

The purpose of this study was to analyze the posterior segment characteristics of the eye in patients who use an SSRI and have no systemic disease other than depression. The main findings of this study were that RNFL thickness and CFT were markedly reduced in individuals with less than a year of SSRI therapy compared to healthy subjects. Conversely, patients with a longer SSRI usage history exhibited no significant disparity when juxtaposed with the control group. Choroidal thickness presented a notable

Table 4. Comparison of the RNFL thickness (µm) in different sectors among the groups

Sector	Group 1a (n=26)	Group 1b (n=26)	Group 2 (n=52)	p
Nasal	64.3±7.2	67.1±7.8	69.5±6.3	0.015
Superior	118.1±11.3	121.7±12.5	126.1±10.1	0.027
Temporal	67.0±8.0	69.8±8.6	72.7±7.2	0.014
Inferior	117.1±10.7	120.4±11.1	124.8±9.5	0.024
Average	91.6±8.8	95.9±9.1	99.7±7.5	0.002

RNFL: Retinal nerve fiber layer, µm: Micrometer.

Table 5. Comparison of the CFT in different points among the groups

Point	Group 1a (n=26)	Group 1b (n=26)	Group 2 (n=52)	p
Fovea center	227.2±18.3	235.1±19.4	244.1±16.2	0.001
1000 µm temporal	241.4±20.2	249.2±21.3	259.1±18.1	0.002
1000 µm nasal	239.2±19.6	247.0±20.7	256.8±17.5	0.003

CFT: Central foveal thickness, µm: Micrometer.

Table 6. Comparison of the choroidal thickness in different points among the groups

Point	Group 1a (n=26)	Group 1b (n=26)	Group 2 (n=52)	p
Fovea center	251.8±35.4	256.7±37.6	288.9±32.1	<0.001
1000 µm temporal	264.5±38.1	268.3±40.3	302.3±35.5	<0.001
1000 µm nasal	260.2±36.7	264.1±39.0	298.1±34.1	<0.001

µm: Micrometer.

decrease in both patient cohorts relative to controls, yet no discernible variance was observed between the patient groups themselves or within each group concerning drug usage duration.

The findings hint at a potential influence of SSRIs on the posterior eye segment, particularly the retina and choroid. SSRIs, primarily prescribed for depression and anxiety disorders, function by amplifying serotonin levels in the central nervous system. Nonetheless, their influence seemingly extends to the eye's posterior segment, encompassing the retina, choroid, optic nerve, and vitreous humor. The serotonergic and anticholinergic pathways of these drugs may underpin the ocular changes noted.[10]

The serotonergic system's modulation stands as a plausible explanation for SSRIs' ocular effects. Serotonin, a multifaceted neurotransmitter, plays a role in mood regulation, cognition, sleep, appetite, pain, and ocular physiology. Its receptors permeate the eye, found in the retina, choroid, ciliary body, iris, and optic nerve. SSRIs inhibit serotonin reuptake at synapses, thereby enhancing its availability and activity within the brain and ocular domain.[11] However, serotonin's ocular impact is intricate, contingent on receptor type, location, density, and interplay with other neurotransmitters, and hormones. Certain receptors, like 5-HT1A and 5-HT2A, may confer neuroprotection to the retina and optic nerve, bolstering retinal ganglion cells and optic nerve axons. In contrast, receptors such as 5-HT1B and 5-HT2C could detrimentally affect the retina and choroid by provoking vasoconstriction, diminishing blood flow and oxygenation to these tissues.[12-14] Thus, SSRIs' retinal and choroidal effects may hinge on the equilibrium between serotonin's neuroprotective and neurotoxic properties, which can vary based on individual characteristics, SSRI usage duration, dosage, and specific SSRI brands.

Historically, sertraline's introduction in 1991 was followed by the identification of sertraline maculopathy a decade later.[8] Case studies have documented varying degrees of maculopathy and visual acuity changes post-SSRI usage, with some instances showing partial reversibility on medication cessation.[15,16] In a case described by Ewe et al.[15] it was stated that bilateral maculopathy developed immediately after starting sertraline in a 23-year-old male patient and that there was a slight improvement in visual symptoms after discontinuation of the drug, but that it did

not return to its previous state. Mason et al.[16] described bilateral bull's eye maculopathy with bilateral visual acuity 20/200 at the age of 14, using sertraline for 1 year, and stated that there was no improvement in retinal pigment epithelium or visual acuity in the 3-year follow-up. These reports align with our findings, suggesting a potential link between SSRI therapy and macular alterations. In a study by Chen et al.,[17] they thought that a possible mechanism of SSRI-induced maculopathy was due to its effect on Phospholipase C. SSRIs can increase 5-HT2AR functionality by increasing the current level of synaptic serotonin and increasing the activation of 5-HT2AR to the activation of Phospholipase C, which then causes an increase in all-trans-retinal (atRAL)-derived NADPH oxidase-mediated intracellular reactive oxygen species so that it may contribute to the pathogenesis of retinal degeneration by causing overproduction and oxidative stress and pathological cell death in the retina.

The choroid, responsible for a substantial portion of ocular blood flow, is integral to the retina's metabolic functions and the optic nerve head and photoreceptors' nourishment.[18] Given its pivotal role, systemic medications, including SSRIs, could influence the choroid and, by extension, the retina and optic nerve's metabolic activities. The eye's susceptibility to drug toxicity, second only to the liver, underscores the importance of understanding medication impacts on ocular health.

Elevated serotonin levels have been implicated in ischemic optic neuropathy. In a study conducted by Lochhead,[7] they reported that elevated serotonin levels may cause ischemic optic neuropathy in patients using SSRI between 6 months and 14 years. From five cases who developed optic neuropathy after SSRI use, they detected anterior ischemic optic neuropathy in one and posterior ischemic optic neuropathy in four, and with the cessation of treatment, the visual loss was permanent except for one. Hayreh[19] stated that high serotonin levels may be associated with ischemic and atherosclerotic diseases. Costagliola et al.[11] described a mechanism for vasospasm in the optic nerve, assuming that increased plasma serotonin levels are a factor in the development of optic nerve perfusion disorders. They reported that with long-term SSRI treatment, multiple transient vasospasms can progressively induce significant ischemic optic neuropathy. While our study did not observe optic nerve disorders, the literature suggests a correlation between high serotonin levels and ischemic conditions. SSRIs' long-term effects could potentially induce significant ischemic optic neuropathy through recurrent transient vasospasms.

SSRIs are a mainstay treatment for depression, but their influence on hemostasis necessitates consideration. SSRIs impede serotonin reuptake in platelets, thereby extending clotting time and elevating bleeding risk. This effect is attributed to serotonin's role in platelet aggregation. Several hemorrhagic complications have been associated with SSRI use, including upper gastrointestinal bleeding and intracranial hemorrhage.[20-24]

Furthermore, studies suggest that SSRIs may deplete platelet serotonin stores with repeated dosing.[25] This phenomenon has led to the proposition of a biphasic effect on bleeding tendency. While the initial SSRI exposure might transiently increase thrombotic risk, chronic use could ultimately lead to heightened bleeding risk due to depleted platelet serotonin stores.[26]

The potential impact of SSRI-induced platelet dysfunction on ocular blood flow warrants investigation. Hemorrhagic complications within the eye, such as uveal effusion, angle-closure glaucoma, and retrobulbar hematoma, have been linked to SSRI use.[21,27,28] Case reports have also described central retinal vein occlusion associated with citalopram, fluoxetine-related retinal vein branch occlusion, and escitalopram-related venous thromboembolism and ischemic stroke.[26,29,30]

These findings suggest a potential link between SSRI-mediated platelet dysfunction and compromised ocular blood flow. Further research is necessary to elucidate the underlying mechanisms and potential clinical implications for patients using SSRIs

Acknowledging the limitations of our study, including the absence of electrophysiological tests, manual choroidal thickness measurements via EDI-OCT, a limited patient cohort, and lack of follow-up assessments, we recognize the need for further research. Future investigations should aim to corroborate our findings, explore the long-term ocular impacts of SSRIs, and refine our understanding of their pharmacological effects on eye health.

Conclusion

Our study sheds light on the discernible alterations in RNFL thickness and CFT associated with short-term SSRI usage, with no significant changes observed in long-term users. The choroidal thinning observed across both patient groups warrants further exploration into SSRIs' systemic and ocular effects. Our research paves the way for future studies to delve deeper into the complex interplay between SSRIs and ocular physiology, ultimately enhancing patient care in the realm of ophthalmology and psychiatry.

Ethics Committee Approval: The study was approved by the university's Ethics Committee (2017/41) and written consent was obtained from all patients and volunteers in accordance with the Declaration of Helsinki of the World Medical Association.

Peer-review: Externally peer-reviewed.

Authorship Contributions: Concept: I.E.Y., K.E.E., N.K.; Design: I.E.Y., K.E.E., N.K.; Supervision: I.E.Y., K.E.E., N.K.; Resource: I.E.Y., K.E.E., N.K.; Materials: K.E.E.; Data Collection and/or Processing: I.E.Y., K.E.E.; Analysis and/or Interpretation: I.E.Y., K.E.E.; Literature Search: I.E.Y., K.E.E., N.K.; Writing: I.E.Y., K.E.E.; Critical Reviews: I.E.Y., K.E.E., N.K.

Conflict of Interest: None declared.

Use of AI for Writing Assistance: Not declared.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Işık E. Depresyon, Tedavi Yaklasımları ve Antidepresan Ilaçlar. Ankara: Gazi Üniversitesi Yayınları; 2006.
2. Duran A. Psikiyatride Ilaçla Tedavi. Türkiye'de sık Karsılasılan Psikiyatrik Hastalıklar Sempozyum Dizisi No: 62; 2008.
3. Hariri A, Ceylan M. Depresyonda ilaç tedavisi: Özgül serotonin gerialim engelleyicileri. [Article in Turkish]. Duygudurum Dizisi 2003;7:339–45.
4. Leonard BE. Principles and practice of psychopharmacotherapy. 3rd edition. P. G. Janicak, J. M. Davis, S. H. Preskorn and F. J. Ayd. Lippincott Williams and Wilkins: Philadelphia, 2001. Pages: 700. ISBN: 0-7817-2794-4. Hum Psychopharmacol Clin Exp 2003;18:317–8.
5. Lejoyeux M Adès J, Léon E. The serotonin syndrome. Focus on Depression and Anxiety 1995;6:4–11.
6. Richa S, Yazbek JC. Ocular adverse effects of common psychotropic agents: A review. CNS Drugs 2010;24:501–26.
7. Lochhead J. SSRI-associated optic neuropathy. Eye (Lond) 2015;29:1233–5.
8. Sener EC, Kiratli H. Presumed sertraline maculopathy. Acta Ophthalmol Scand 2001;79:428–30.
9. Kocer E, Kocer A, Ozsutcu M, Dursun AE, Krpnar I. Dry eye related to commonly used new antidepressants. J Clin Psychopharmacol 2015;35:411–3.
10. Bauer M, Monz BU, Montejo AL, Quail D, Dantchev N, Demyttenaere K, et al. Prescribing patterns of antidepressants in Europe: Results from the Factors Influencing Depression Endpoints Research (FINDER) study. Eur Psychiatry 2008;23:66–73.
11. Costagliola C, Parmeggiani F, Sebastiani A. SSRIs and intraocular pressure modifications: Evidence, therapeutic implications and possible mechanisms. CNS Drugs 2004;18:475–84.
12. Masson J. Serotonin in retina. Biochimie 2019;161:51–5.
13. Palacios JM, Pazos A, Hoyer D. A short history of the 5-HT2C receptor: From the choroid plexus to depression, obesity and addiction treatment. Psychopharmacology (Berl) 2017;234:1395–418.
14. Alfaro CL, Lam YW, Simpson J, Ereshefsky L. CYP2D6 status of extensive metabolizers after multiple-dose fluoxetine, fluvoxamine, paroxetine, or sertraline. J Clin Psychopharmacol 1999;19:155–63.
15. Ewe SY, Abell RG, Vote BJ. Bilateral maculopathy associated with sertraline. Australas Psychiatry 2014;22:573–5.
16. Mason JO 3rd, Patel SA. Bull'S eye maculopathy in a patient taking sertraline. Retin Cases Brief Rep 2015;9:131–3.
17. Chen Y, Okano K, Maeda T, Chauhan V, Golczak M, Maeda A, et al. Mechanism of all-trans-retinal toxicity with implications for stargardt disease and age-related macular degeneration. J Biol Chem 2012;287:5059–69.
18. Nickla DL, Wallman J. The multifunctional choroid. Prog Retin Eye Res 2010;29:144–68.
19. Hayreh SS. Retinal and optic nerve head ischemic disorders and atherosclerosis: Role of serotonin. Prog Retin Eye Res 1999;18:191–221.
20. Turner MS, May DB, Arthur RR, Xiong GL. Clinical impact of selective serotonin reuptake inhibitors therapy with bleeding risks. J Intern Med 2007;261:205–13.
21. Zelefsky JR, Fine HF, Rubinstein VJ, Hsu IS, Finger PT. Escitalopram-induced uveal effusions and bilateral angle closure glaucoma. Am J Ophthalmol 2006;141:1144–7.
22. Van Walraven C, Mamdani MM, Wells PS, Williams JI. Inhibition of serotonin reuptake by antidepressants and upper gastrointestinal bleeding in elderly patients: Retrospective cohort study. BMJ 2001;323:655–8.
23. Loke YK, Trivedi AN, Singh S. Meta-analysis: Gastrointestinal bleeding due to interaction between selective serotonin uptake inhibitors and non-steroidal anti-inflammatory drugs. Aliment Pharmacol Ther 2008;27:31–40.
24. De Abajo FJ, Jick H, Derby L, Jick S, Schmitz S. Intracranial haemorrhage and use of selective serotonin reuptake inhibitors. Br J Clin Pharmacol 2000;50:43–7.
25. Sauer WH, Berlin JA, Kimmel SE. Selective serotonin reuptake inhibitors and myocardial infarction. Circulation 2001;104:1894–8.
26. Hardisty AD, Hemmerdinger CM, Quah SA. Citalopram-associated central retinal vein occlusion. Int Ophthalmol 2009;29:303–4.
27. Arias Palomero A, Infantes Molina EJ, López Arroquia E, Riveira Villalobos L, López Mondéjar E, González Del Valle F. Uveal effusion induced by escitalopram. Arch Soc Esp Oftalmol 2015;90:327–30.
28. Van Cann EM, Koole R. Retrobulbar hematoma associated with selective serotonin reuptake inhibitor: A case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108:e1–2.
29. Knox Cartwright NE, Smith P, Tole DM. Branch retinal vein occlusion and fluoxetine. Ann Ophthalmol (Skokie) 2007;39:253–4.
30. Kurne A, Ertugrul A, Anil Yagcioglu AE, Yazici KM. Venous thromboembolism and escitalopram. Gen Hosp Psychiatry 2004;26:481–3.