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***Observational Study***

**Impact of coronary artery bypass grafting surgery on the chorioretinal biomicroscopic characteristics**

Shahriari M *et al*. Chorioretinal characteristics after CABG

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**Abstract**

BACKGROUND

Most patients with cardiovascular disorders suffer from coronary artery diseases, which can be treated successfully using coronary artery bypass grafting (CABG). One of the unpleasant events following CABG is postoperative vision loss (POVL). Vulnerability of retinal vessels to hemodynamic changes, an expectable event following CABG, may contribute to the development of POVL, which might be associated with the changes in the choroidal and retinal structures.

AIM

To investigate postoperative changes in chorioretinal and peripapillary nerve fiber layer (NFL) thickness, and progression of diabetic and hypertensive retinopathy after CABG.

METHODS

In this prospective, cross-sectional study, 49 eyes in 25 candidates for CABG underwent both ophthalmic and cardiovascular examinations within 6 mo prior to and 9 mo after surgery.

RESULTS

Among the study participants, 56% were male with a mean age of 62.84 years ± 10.49 years (range 33–80 years). Diabetes mellitus was observed in eight participants (32%). None of the patients suffered from postoperative anterior or posterior ischemic optic neuropathy, central retinal artery occlusion, and cortical blindness. The mean value of the preoperative best corrected visual acuity was 0.11 ± 0.10 logMAR (range, 0–0.4), which worsened to 0.15 ± 0.08 logMAR (range, 0–0.4) after CABG (*P* = 0.031). No significant difference was observed between the pre- and postsurgical choroidal (*P* = 0.853) and macular (*P* = 0.507) thickness, NFL thickness in the subfoveal (*P* > 0.999) and peripapillary areas (*P* = 0.659), as well as the severity of diabetic and hypertensive retinopathy.

CONCLUSION

CABG may reduce visual acuity without affecting ocular structures. Postoperative vision reduction might be attributable to molecular or cellular variations, changes in visual pathway function, or central nervous system.

**Key Words:** Coronary artery bypass grafting; Nerve fiber layer; Diabetic retinopathy; Hypertensive retinopathy

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**Core Tip:** This was a prospective cohort study investigating the effect of coronary artery bypass grafting (CABG) on the chorioretinal and peripapillary nerve fiber layer thicknesses, as well as the progression of diabetic and hypertensive retinopathy. CABG may significantly reduce the visual acuity without impacting on the ocular structures.

**INTRODUCTION**

Nowadays, cardiovascular diseases are considered one of the most common systemic disorders potentially leading to death worldwide[1]. The mortality rate of cardiovascular diseases is 6.4 per 10 000 individuals in Iran[2]. Additionally, it has been shown that the comorbidity of other systemic diseases such as diabetes, hyperlipidemia, and hypertension could be influential on the increasing risk of cardiovascular diseases[3,4].

Most patients with cardiovascular disorders suffer from coronary artery diseases, which can be treated successfully using coronary artery bypass grafting (CABG). Although this type of surgery was first described in 1968, currently it applies extensively to nearly 60% of patients with coronary artery ischemia[5-8]. An improvement in the quality of life of patients who undergo CABG has been reported. However, one of the unpleasant events following CABG is postoperative vision loss (POVL)[9,10].The overall incidence rate of POVL in the USA is ~1 in 60 000 to 125 000 cases. This event is more frequent following spinal and cardiac surgery[11-14]. Vulnerability of retinal vessels to hemodynamic changes, an expected event following CABG, may contribute to the development of POVL[15,16]. The retinal and choroidal vascular systems have several characteristics making them vulnerable to procedures like CABG. First, there is no anastomosis in the retinal and choroidal vascular systems. The inner two thirds of retinal tissue is supplied by the central retinal artery and the rest of the retinal tissue is nourished by the choroidal vascular network. The retinal circulation relies on local vascular control mechanisms. The choroidal neurons, sympathetic, parasympathetic and sensory innervation, and the factors secreted by the endothelium control the blood flow of the retina[17].

Presumably, the hemodynamic dysfunction due to surgery and postoperative ischemia can result in the activation of hypoxic/ischemic-related biochemical pathways[16,18,19]. These changes could have short- and long-term effects on the sensitive structures to hypoperfusion and ischemia, a condition similar to other vascular diseases like ocular ischemic syndrome or diabetic retinopathy[18,19]. The ocular complications might be associated with the changes in the choroidal, retinal, and nerve fiber layer (NFL) thicknesses caused by either short-term edematous processes leading to activation of the hypoxic biomechanical pathways or structural changes due to the long-term decrease in blood flow and nutritional supply of the eye[20].

Aging can markedly decrease choroidal thickness. Moreover, several systemic and ocular diseases can affect choroidal thickness. For example, hypertensive retinopathy and hypercholesterolemia (without coronary artery disease (CAD)) increase the choroidal thickness. However, cigarette smoking, ocular ischemic syndrome, chronic heart failure, and systemic essential hypertension have the opposite effect[21,22]. Some drugs such as corticosteroids and atorvastatin can change choroidal thickness. Also, carotid stenosis can affect choroidal thickness[21,23]. One study showed that carotid stenosis leads to subfoveal choroidal thinning[23]. Previous studies showed that patients with CAD have lower choroidal thickness, which can be a prognostic biomarker of heart disease[22].It seems that in addition to decreased retinal and choroidal thickness, attenuated vascular density of the central retinal region may be a prognostic factor for CAD[24,25]. It can be stated that the main leading cause of the ocular complications following CABG could come from the decreased rate of blood flow to the ocular structures and visual pathways that might be associated with the consequent atrophic changes in the retina, choroid, and optic nerve[26-28]. Therefore, the current study aimed to investigate the postoperative changes of the chorioretinal and peripapillary NFL thicknesses, as well as the progression of diabetic and hypertensive retinopathy in patients undergoing CABG.

**MATERIALS AND METHODS**

***Study population***

In this prospective, cross-sectional study, 58 candidates for CABG from September 2019 to January 2022 were consecutively recruited. Patients with pathological conditions affecting NFL thickness, such as optic disc drusen, retinal vein occlusion, and glaucoma, were excluded (*n* = 12). Also, 21 patients were excluded due to having other ocular pathologies that required urgent therapeutic interventions (*n* = 4), vitreoretinal injection (*n* = 4), diabetic maculopathy (*n* = 4), and missing follow-up data (*n* = 9). Finally, this study was conducted on 49 in 25 candidates for CABG.

***Ethical consideration***

This study was approved by the Ethics Committee of the Ophthalmic Research Center, Shahid Beheshti university of Medical Sciences (approval number IR.SBMU.MSP.REC.1398.224). The study details were explained and a written informed consent letter was signed by all participants.

***Data collection***

Initially, all the patients were interviewed to record baseline information of their age, history of smoking, systemic diseases including hypertension, diabetes, hyperlipidemia, and pulmonary diseases, as well as medication history. The clinical history of the cardiovascular examinations, including ejection fraction and history of previous transient ischemic attack or cerebrovascular accident, was recorded by an experienced cardiologist. Intraoperative information including on- or off-pump CABG, duration of pump usage, number of grafted coronary vessels, and duration of CABG were collected.

Additionally, a comprehensive ophthalmic examination including assessment of the best corrected visual acuity (BCVA) by the Snellen E chart and measurement of intraocular pressure using the Goldmann tonometer were conducted before and after CABG by a single ophthalmologist. The visual acuities were converted to logMAR for statistical analysis. The biomicroscopy and fundus examinations were conducted on a dilated pupil to identify other ocular pathologies. The diabetic retinopathy was staged by the standard classification of Early Treatment Diabetic Retinopathy Study system, and disease progression was defined as progression to the higher stages. Hypertensive retinopathy was classified according to the Modified Scheie Classification system and disease progression was defined as the progression to the higher stages.

The thickness of NFL, macular subfoveal area, and vascular calibration were measured by spectral-domain optical coherence tomography (SD-OCT; Spectralis, Heidelberg, Germany). In addition, the choroidal thickness measurement was conducted manually in the subfoveal area using Enhanced depth imaging-OCT (EDI-OCT; Spectralis). These procedures were repeated three times for each participant and the mean of the findings was applied in the final analysis. The mean value of the four quadrants of the optic disc was considered as the final peripapillary NFL. The macular NFL was estimated by the measured parameters in EDI-OCT. Vascular calibration of the retina was performed manually based on the infrared images of the optic disc using three main arteries or veins that located within the one half to one-disc diameter from the disc margin. Afterwards, the mean values of the arteries or veins were considered as the final measurement.

All the examinations and imaging were conducted within 6 mo prior to CABG and repeated 9 mo after surgery.

***Statistical analysis***

Frequency (percentage), mean ± SD, median, and range were used to describe the data. After checking the normality of the distribution of variables using the Kolmogorov–Smirnov test, paired *t* test was used to compare the variables before and after CABG. To compare two eyes in one person, we utilized generalized estimating equation analysis. All statistical analysis was performed by IBM SPSS Statistics for Windows version 25.0 (Armonk, NY, USA). *P* < 0.05 was considered statistically significant.

**RESULTS**

In the current study, 49 eyes in 25 candidates for CABG were investigated. Among the study participants, 56% were male and the mean age was 62.84 ± 10.49 years (range 33–80). Diabetes mellitus was observed in eight participants (32%). Out of these, three cases had mild nonproliferative diabetic retinopathy (NPDR) and one had moderate NPDR. Status of diabetic retinopathy remained stable after surgery. Thirteen patients (52.0%) mentioned a history of systemic hypertension, of whom, four showed grade I hypertensive retinopathy. No changes were detected in the grading of hypertensive retinopathy after surgery. None of the patients suffered from postoperative anterior or posterior ischemic optic neuropathy, central retinal artery occlusion, and cortical blindness (Table 1).

The mean value of the preoperative BCVA was 0.11 ± 0.10 logMAR (range, 0–0.4), which worsened to 0.15 ± 0.08 logMAR (range, 0–0.4) after CABG (*P* = 0.031) (Table 2).

No significant changes were observed in the comparison of pre- and postoperative subfoveal choroidal thickness (pre: 283.49 µm ± 42.79 µm *vs* post: 283.29 µm ± 43.21 µm, *P* = 0.853); macular thickness (pre: 267.92 µm ± 31.64 µm *vs* post: 267.33 µm ± 31.19 µm, *P* = 0.507); macular NFL (pre: 12.35 µm ± 2.30 µm *vs* post: 12.35 µm ± 2.64 µm, *P* > 0.999); peripapillary NFL (pre: 105.1 µm ± 12.36 µm *vs* post: 104.98 µm ± 12.84 µm, *P* = 0.659); venous caliber (pre: 101.13 µm ± 9.2 µm *vs* post: 100.96 µm ± 9.83 µm, *P* = 0.673); and arterial caliber (pre: 79.42 µm ± 7.62 µm *vs* post: 79.39 µm ± 7.94 µm, *P* = 0.923) (Table 3).

In the present study, separate analysis was conducted on eight patients with diabetes mellitus and no significant changes in BCVA and ocular structures were detected after CABG (Table 4).

Furthermore, additional analysis on the patients with POVL (11 patients) showed significant macular thickening after surgery, without any changes in the other ocular parameters (Table 5).

No significant difference was detected in smoking, systemic hypertension, IOP, and medication between patients with and without vision loss. Additionally, no significant difference was found in the involved vessels, as well as the ejection fraction between these two groups.

**DISCUSSION**

The current study was conducted on 49 eyes in 25 candidates for CABG, in order to identify the changes in the chorioretinal biomicroscopic characteristics. We observed that the mean BCVA worsened after CABG, while no significant changes were detected in the other study parameters. The lack of chorioretinal changes after CABG could be because all patients were operated upon by off-pump CABG. Comparing the degree of inflammatory response with the levels of proinflammatory cytokines and inflammatory markers in both techniques, it was revealed that the release of interleukin-8, interleukin-6, and tumor necrosis factor receptors 1 and 2 were higher in the on-pump CABG (ONCAB) group than the off-pump CABG (OFFCAB) group[29]. Kocamaz *et al*[22] reported that the main cause of the postoperative vision decrease following CABG could be attributed to ischemic optic neuropathy and vascular occlusion that occurred secondary to emboli. Many studies using transcranial Doppler have shown higher rates of cerebral vessel embolization in ONCAB compared with OFFCAB [29]. Similarly, Ala-Kauhaluoma *et al*[23] did not find any association between ischemic optic neuropathy and the other risk factors including systemic hypertension, CAD, diabetes mellitus and stroke. Furthermore, Pekel *et al*[16] observed no chorioretinal change after ONCAB.

In the present study, POVL could not be attributed to ocular changes due to lack of significant postoperative changes in the peripapillary NFL thickness, venous and arterial caliber, ischemic optic disc (anterior and posterior optic neuropathy), and embolic changes. Along with our findings, Williams *et al*[15] observed no significant changes between the patients undergoing CABG and controls regarding the ocular parameters, including peripapillary RNFL, ocular pulse amplitude, retinal vascular caliber and macular thickness. They concluded that these changes were not identifiable even after the long-term follow-up[15]. However, we found the same findings in the short-term follow-up after CABG. Conversely, Buyukates *et al*[27] reported that the surgical procedure of cardiopulmonary bypass can result in a transient decrease in retinal NFL thickness.

In this study, we conducted additional investigation on patients with diabetes mellitus, since it has been reported as a potential risk factor that could have affected our study outcomes. While our findings on diabetic patients were the same as nondiabetic participants, there is a debate about the effect of diabetes mellitus on choroidal thickness. In several studies, an increase in choroidal thickness has been detected in diabetic patients[25]. However, in some other studies, an opposite effect has been reported[17].

Our investigation of patients with vision loss showed that they had significant thickening of the macula, which might be illustrative of a transient hypoxic or ischemic process that happened during or after CABG. Furthermore, the POVL could be attributed to molecular or cellular variations or changes in visual pathways’ function or central nervous system. While this reduction could not be due to the structural changes that happened after CABG surgery.

This was a case series with a limited number of participants due to the low referral rate of our patients secondary to the COVID-19 pandemic. Another limitation of the present study could be related to the lack of consideration of factors such as duration of surgery and intraoperative hemodynamic conditions. Also, no investigation was conducted on the effects of intraoperative perfusion pump usage since all of the patients were operated by OFFCAB. Lastly, optical coherence tomography angiography can act as a useful imaging modality in such studies. However, at the time of study, this method was not available in our center. So, running larger studies with more evaluations such as OCT-A is recommended.

**CONCLUSION**

We evaluated the chorioretinal biomicroscopic changes in patients undergoing CABG. Our findings showed that these patients may experience POVL with no changes in chorioretinal structures. POVL could be attributed to molecular or cellular variations or changes in the visual pathway function or central nervous system.

**ARTICLE HIGHLIGHTS**

***Research background***

Coronary artery bypass grafting (CABG) is currently used in most patients with coronary artery diseases. One of the unfavorable reported events following CABG is postoperative vision loss.

***Research motivation***

Ocular structures are vulnerable to hemodynamic changes, which are expected during and after CABG. Changes in choroidal and retinal structures can be the cause of this complication.

***Research objectives***

To assess the changes in choroidal and retinal structures after CABG.

***Research methods***

A total of 49 eyes in 25 candidates for CABG underwent comprehensive ophthalmic and cardiovascular examination before and after surgery.

***Research results***

A decrease in visual acuity was detected, while changes in choroidal and retinal structures were not significant.

***Research conclusions***

CABG may lead to visual acuity reduction without affecting the ocular structures. The possibility of cellular or central nervous system damage should be considered.

***Research perspectives***

Larger studies with further evaluation such as optical coherence tomography angiography are required in this regard.

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**Footnotes**

**Institutional review board statement:** This study was reviewed and approved by the Ethics Committee of the Ophthalmic Research Center, Shahid Beheshti university of Medical Sciences (Approval No. IR.SBMU.MSP.REC.1398.224).

**Informed consent statement:** All study participants, or their legal guardian, provided informed written consent prior to study enrollment.

**Conflict-of-interest statement:** All the authors report no relevant conflicts of interest for this article.

**Data sharing statement:** There is no additional data available.

**STROBE statement:** The authors have read the STROBE Statement—checklist of items, and the manuscript was prepared and revised according to the STROBE Statement—checklist of items.

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**Table 1 Demographic characteristics of the participants,** ***n* (%)**

|  |  |  |
| --- | --- | --- |
| **Item** | | **Value** |
| Sex | Male | 14 (56.0) |
|  | Female | 11 (44.0) |
| Diabetic | No | 17 (68.0) |
|  | Yes | 8 (32.0) |
| Systemic hypertension | No | 12 (48.0) |
|  | Yes | 13 (52.0) |
|  |  |  |
| Age, yr | Mean ± SD | 62.84 ± 10.49 |
|  | Median (range) | 64 (33-80) |

**Table 2 Changes in best-corrected visual acuity after coronary artery bypass grafting**

|  |  |  |
| --- | --- | --- |
|  | **Mean ± SD** | **Median (range)** |
| Pre-surgery BCVA (decimal) | 0.80 ± 0.16 | 0.8 (0.4-1.0) |
| Post-surgery BCVA (decimal) | 0.70 ± 0.29 | 0.8 (0.4-1.0) |
| *P* value1 | 0.037 |  |
| Pre-surgery BCVA (logMAR) | 0.11 ± 0.10 | 0.1 (0-0.4) |
| Post-surgery BCVA (logMAR) | 0.15 ± 0.08 | 0.1 (0-0.4) |
| *P* value1 | 0.031 |  |

1Paired *t*-test.

BVCA: Best-corrected visual acuity.

**Table 3 Comparison of choroidal and macular thicknesses and macular and prepapillary nerve fiber layer thicknesses before and after coronary artery bypass grafting**

|  |  |  |
| --- | --- | --- |
|  | **Mean ± SD** | **Median (range)** |
| Preoperative choroidal thickness | 283.49 ± 42.79 | 289 (183-366) |
| Postoperative choroidal thickness | 283.29 ± 43.21 | 291 (190-359) |
| *P* value1 | 0.853 |  |
| Preoperative macular thickness | 267.92 ± 31.64 | 263 (221-426) |
| Postoperative macular thickness | 267.33 ± 31.19 | 261 (218-420) |
| *P* value1 | 0.507 |  |
| Preoperative macular NFL thickness | 12.35 ± 2.30 | 12 (8-18) |
| Postoperative macular NFL thickness | 12.35 ± 2.64 | 13 (7-19) |
| *P* value1 | > 0.999 |  |
| Preoperative prepapillary NFL thickness | 105.10 ± 12.36 | 103 (79-136) |
| Preoperative prepapillary NFL thickness | 104.98 ± 12.84 | 101 (76-137) |
| *P* value1 | 0.659 |  |
| Preoperative venous caliber | 101.13 ± 9.20 | 100.6 (85-121) |
| Postoperative venous caliber | 100.96 ± 9.83 | 102.3 (85.0-123.2) |
| *P* value1 | 0.673 |  |
| Preoperative arterial caliber | 79.42 ± 7.62 | 79 (66.3-98.0) |
| Postoperative arterial caliber | 79.39 ± 7.94 | 79 (65.4-100.2) |
| *P* value1 | 0.923 |  |

1Paired *t*-test.

NFL: Nerve fiber layer.

**Table 4 Comparison of best-corrected visual acuity, choroidal and macular thicknesses, macular and prepapillary** **nerve fiber layer thicknesses, arterial and venous caliber before and after coronary artery bypass grafting in diabetic patients**

|  |  |  |  |
| --- | --- | --- | --- |
|  | **Mean ± SD** | **Median (range)** | ***P* value1** |
| Preoperative BCVA (decimal) | 0.75 ± 0.12 | 0.75 (0.50, 0.90) | > 0.999 |
| Postoperative BCVA (decimal) | 0.75 ± 0.13 | 0.75 (0.50, 1.00) | |
| Preoperative BCVA (logMAR) | 0.13 ± 0.07 | 0.13 (0.05, 0.30) | > 0.999 |
| Postoperative BCVA (logMAR) | 0.13 ± 0.07 | 0.13 (0, 0.30) | |
| Preoperative choroidal thickness | 297 ± 51.73 | 314.5 (183.0, 343.0) | 0.532 |
| Postoperative choroidal thickness | 298.44 ± 53.61 | 320 (190, 346) | |
| Preoperative macular thickness | 271.56 ± 43.36 | 262 (240, 426) | 0.126 |
| Postoperative macular thickness | 273.50 ± 41.83 | 262 (237, 420) | |
| Preoperative macular NFL thickness | 12.13 ± 1.67 | 12 (8, 14) | 0.203 |
| Postoperative macular NFL thickness | 12.56 ± 2.37 | 12.5 (9.0, 17.0) | |
| Preoperative prepapillary NFL thickness | 105.50 ± 13.24 | 98.5 (93.0, 127.0) | 0.060 |
| Postoperative prepapillary NFL thickness | 106.19 ± 13.99 | 99.5 (92.0, 129.0) | |
| Preoperative venous caliber | 102.30 ± 8.51 | 102.6 (88.3, 115.3) | 0.415 |
| Postoperative venous caliber | 101.74 ± 9.73 | 103.7 (85.0, 114.7) | |
| Preoperative arterial caliber | 77.56 ± 7.68 | 77.8 (68.0, 95.6) | 0.363 |
| Postoperative arterial caliber | 77.03 ± 8.27 | 76.45 (65.40, 91.60) | |

1Paired *t*-test.

BVCA: Best-corrected visual acuity; NFL: Nerve fiber layer.

**Table 5 Comparison of best-corrected visual acuity, choroidal and macular thicknesses, macular and prepapillary nerve fiber layer thicknesses, arterial and venous caliber before and after coronary artery bypass grafting in patients with vision loss**

|  |  |  |  |
| --- | --- | --- | --- |
|  | **Mean ± SD** | **Median (range)** | ***P* value1** |
| Preoperative BCVA (decimal) | 0.75 ± 0.16 | 0.8 (0.5, 0.9) | 0.002 |
| Postoperative BCVA (decimal) | 0.64 ± 0.17 | 0.7 (0.4, 0.8) | |
| Preoperative BCVA (logMAR) | 0.13 ± 0.10 | 0.10 (0.05, 0.30) | 0.003 |
| Postoperative BCVA (logMAR) | 0.21 ± 0.13 | 0.15 (0.10, 0.40) | |
| Preoperative choroidal thickness | 290.36 ± 37.70 | 304 (212, 343) | 0.964 |
| Postoperative choroidal thickness | 290.27 ± 40.06 | 300 (218, 345) | |
| Preoperative macular thickness | 262.18 ± 15.31 | 261 (235, 284) | 0.035 |
| Postoperative macular thickness | 264.73 ± 15.77 | 260 (233, 286) | |
| Preoperative macular NFL thickness | 12.64 ± 2.11 | 12 (10, 18) | 0.903 |
| Postoperative macular NFL thickness | 12.64 ± 2.66 | 13 (10, 19) | |
| Preoperative prepapillary NFL thickness | 106.55 ± 11.12 | 110 (89, 125) | > 0.999 |
| Postoperative prepapillary NFL thickness | 106.64 ± 11.75 | 112 (89, 126) | |
| Preoperative venous caliber | 97.30 ± 9.03 | 96.0 (87.0, 110.6) | 0.228 |
| Postoperative venous caliber | 96.60 ± 10.05 | 95.0 (85.1, 109.0) | |
| Preoperative arterial caliber | 78.45 ± 6.34 | 79.6 (66.3, 86.3) | 0.689 |
| Postoperative arterial caliber | 77.98 ± 6.98 | 78.3 (66.0, 91.0) | |

1Paired *t* test.

BVCA: Best-corrected visual acuity; NFL: Nerve fiber layer.