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Ocular Complications in Retinal Vein Occlusion

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Abstract

Purpose: To investigate the factors that are responsible for the ocular complications in retinal vein occlusion.

Methods: A total of 106 patients with retinal vein occlusion (RVO) were enrolled in this retrospective study. Detailed ocular and medical history, ophthalmologic examination including best corrected visual acuity (BCVA), slit lamp examination of the anterior segment, intraocular pressure, relative afferent pupillary defect, visual field test, and fundus evaluation was done. Fundus fluorescein angiography (FA) was used to evaluate the extent of the vascular occlusion, the degree of ischemia, the presence or absence of perivenous staining, to localize leaking micro aneurysms, distinguish collateral vessels, and the type of macular edema (ischemic vs. non-ischemic). The classification regarding non-ischemic and ischemic central retinal vein occlusion (CRVO) and also major and peripheric branch retinal vein occlusion (BRVO) was performed by using FA and ophthalmoscopy. All patients were followed up for 1 year at 3-month intervals from the first examination. For each visit, visual prognosis, the inner retinal ischemia signs of anterior and posterior neovascularization, vision treated ocular complications in the early and late period were evaluated.

Results: A 118 eyes of the 106 patients consisting of 62 men and 44 women, with a mean age of 61.5 ± 12.3 years and 59.0 ± 9.8 years respectively. There were 37(31.4%) eyes with CRVO, and 81 (68.6%) eyes with BRVO. Of the 37 CRVO eyes were ischemic, 16 were non- ischemic and 4 were conversion of non-ischemic into ischemic CRVO. Of the 81 BRVO eyes, 64 (54.2%) were major and 17(14.4%) were peripheral BRVO. Systemic hypertension and diabetes mellitus were observed with the rates of 70.8% and 5.7% respectively in 106 RVO patients. The most prevalent ocular risk factors were primary open angle glaucoma was found in 14(37.8%) eyes with CRVO and ocular hypertension was found in 3(2.9%) eyes with BRVO. The initial visual acuities ranged from 20/40, 20/50-20/100, and 20/200. A patient with ischemic CRVO and conversion of non-ischemic into ischemic CRVO final visual acuity was 20/200 or worse due to chronic cystoid macular edema (CME) and optic atrophy. In 64 major BRVO eyes 54.7% of them recovering vision to 20/40 or better spontaneously. Complications included neovascular glaucoma (NVG), seen in 9 (52.9%) eyes of ischemic, and in 3(4.7%) eyes of major BRVO. Retinal neovascularization was observed in 15 (88.2%) eyes of ischemic CRVO, 4 (100%) eyes of conversion of non-ischemic into ischemic CRVO and 15(23.4%) eyes with major BRVO. Late changes seen in the macular area was cystoid macular edema was CME observed in 8 (47%) eyes of ischemic CRVO, 3(18.8%) eyes of non-ischemic CRVO and 3(75%) eyes conversion of non-ischemic into ischemic CRVO eyes. In BRVO CME was seen in 32 (50%) of the major BRVO eyes.

Conclusion: The two main vision treated factors in RVO are known as macular edema and neovascularization. This can be reduced by modifying known risk factors and early appropriate therapy.

Keywords: Retinal vein occlusion; Ischemia; Complications

Introduction

Central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO) are the two main types of ocular vascular occlusive disorders which are the most common causes of visual disability in the middle-aged and elderly person [1]. Systemic hypertension, diabetes mellitus and hyperlipidemia are the main associated factors for any form of retinal vein occlusion (RVO). The exact pathogenesis of RVO remains unclear. Trombotic events, external compression, or vessel wall pathology are the factors that are responsible from vascular occlusion [2,3]. This condition may be due to a combination of three systemic changes known as Virchow's triad: hemodynamic changes (venous stasis), degenerative changes of the vessel wall and blood hypercoagulability. RVO is classified based on anatomic location and degree of retinal ischemia [4]. It is believed that compression externally on the wall of the retinal vein in the lamina cribrosa (CRVO) or at an arterio-venous crossing (BRVO) by the adjacent artery [5-7].

According to the Central Vein Occlusion Study (CVOS), these subtypes can be further classified as ischemic if fluorescein angiography (FA) reveals greater than 10 disc diameters of retinal capillary no perfusion, as perfused if fewer than 10 disc diameters of ischemia are present, or as indeterminate if an accurate determination of the degree of non-perfusion cannot be estimated due to significant retinal haemorrhage. These two forms have different pathogenesis, clinical features, prognosis and management. Non-ischemic CRVO is the most common type, accounting for about 75% of cases [2]. The characteristics of non-ischemic CRVO include good visual acuity (VA), a mild or no afferent pupillary defect, and mild visual changes. Non-ischemic CRVO can also be referred to as partial, perfused, or venous stasis retinopathy. There is no capillary perfusion defect on FA.

Ischemic CRVO can be primary or progression of a non-ischemic CRVO, although progression is not common. Ischemic CRVO has a much worse visual prognosis and accounts for about 30% of cases. Other names for ischemic CRVO include complete, no perfused, or haemorrhagic retinopathy. Major BRVO can be asymptomatic or with visual blurring usually involving the sector of visual field corresponding to the area of the retina involved. In macular BRVO, there is always central visual disturbance with normal peripheral vision. 50-60% of eyes have been reported to have a final VA of 20/40 or better even without any treatment. CRVO leads to hypoxia in the retinal tissues and the subsequent release of vascular endothelial growth factor (VEGF) and inflammatory mediators. Complications of this include macular edema, vitreous haemorrhage, and neovascular glaucoma (NVG). Iris neovascularization develops in 2/3 of cases of ischemic CRVO. When iris neovascularization develops, 1/3 of these develop NVG. A total of 10% of these cases occur in combination with branch retinal artery occlusion. Macular edema (ME) can lead to significant vision loss in patients with CRVO. Intravitreal anti-VEGF injections have been used to decrease the edema and improve visual acuity. RVO can result in permanent vision impairment or blindness. Early recognition and prompt treatment are key to preserving vision and achieving good outcomes. In this article we review the epidemiology, risk factors, and clinical features of RVO [7].

Methods

The medical records of 106 patients with CRVO and BRVO were reviewed retrospectively. Patients with history of ocular surgery or trauma were not included in this study. The study protocol adhered to the tenets of Declaration of Helsinki and was approved by the ethics committee. Fully informed written concert form from patients were taken. All included patients had detailed ocular and medical history, laboratory workup included a complete blood count, sedimentation rate, fasting blood sugar, lipid profile, ophthalmologic examination including BCVA, slit lamp examination of the anterior segment, afferent pupillary defect, intraocular pressure, visual field testing with Goldman perimeter, fundus evaluation by direct and indirect ophthalmoscopy, and, if required, by contact lens, and fundus fluorescein angiography were done. All patients were followed up for 1 year at 3- month intervals from the first examination. For each visit, visual prognosis, vision treated ocular complications, retinal haemorrhages at various locations in the retina, macular edema, optic disc edema, cotton-wool spots, other fundus parameters; the presence of epiretinal membrane, macular retinal pigment epithelial degeneration, macular hole, retinal venous sheathing, optic disc collaterals, optic disc pallor were all recorded. Patients were classified as ischemic or non-ischemic CRVO and major and peripheric BRVO by using FA and ophthalmoscopy. When the retinopathy allowed a sufficient angiogram, colour fundus photography and fundus fluorescein angiography were done using a 60 degree Canon fundus camera. Images were taken approximately one minute (arteriovenous phase) and 4-5min (late venous phase) after intravenous injection of Sodyum Fluorescein 10%, 5 ml vial. We evaluated the presence or absence of fluorescein staining, perivenous fluorescein staining, microaneurysms, retinal capillary obliteration, retinal capillary foveal arcade intact or broken, and arteriovenous filling time (in seconds) in the fovea, perifovea, macula, the rest of the posterior pole (outside the vascular arcades) and peripheral retina.

Results

A 118 eyes of 106 patients with RVO, 62(58.5%) men and 44(41.5%) women, were examined in this retrospective study.

The mean age for men was 61.5±12.3 years (range, 29-85 years), and for women was 59.0±9.8 (range, 42-83 years). Among the study subject's hypertension was found 78.8% in CRVO and 66.7% in BRVO. Diabetes mellitus was found in 3.0% of 117 CRVO and 9.3% of BRVO patients. Trauma, tuberculosis, migraine were observed in BRVO patients with a rate of 4%, 1.3%, and 1.3% respectively. 64.5% of men with RVO were smokers and taking alcohol for a long period of time. That was only seen in 4.6% of women. 9.1% of women were using oral contraceptive. Hyperlipidemia and hypercholesterolemia were found in 60.6%, 24.2% in CRVO patients and 38%, 16.2% in BRVO patients with respectively. The most prevalent ocular risk factors for CRVO were primary open angle glaucoma and ocular hypertension, which were found in 14 (37.8%) of 37 patients with CRVO (12 had open angle glaucoma, 2 had ocular hypertension) and in (3.7%) of 81 patients with BRVO had ocular hypertension. Left eye involvement was seen in 23 (62.2%) of 37 CRVO eyes, and 43(53.1%) of 81 BRVO eyes. Hypermetrophy was found in 23 (62.2%) eyes with CRVO, and 57(70.4%) eyes with BRVO (Table 1).

Table 1: *Systemic and ocular conditions among the subjects with RVO.*

	CRVO	BRVO
Age	61.5±12.3	59.0±9.8
Female	11(10.4%)	33(37.7%)
Male	22(20.8%)	40(31.1%)
Systemic Condition		
Hypertension	78.80%	66.70%
Diabetes mellitus	3.00%	9.30%
Hypertriglyceridemia	60.60%	38%
Hypercholesterolomia	24.20%	16.20%
Ocular Factors		
Open angle glaucoma	12(32.4%)	-
Ocular hypertension	2(5.4%)	3(3.7%)
Laterality		
Right eye	14(37.8%)	38(46.9%)
Left eye	23(62.2%)	43(53.1%)
Refractive error		
Myopia	14(37.8%)	24(29.6%)
Hypermetropia	23(62.2%)	57(70.4%)

There were 25 (67.5%) eyes with CRVO and 45(55.6%) eyes with BRVO seen within the first 90 days after the onset of the disease and 12 (32.4%) eyes with CRVO and 36 (44.4%) eyes with BRVO seen within 90 to 365 days. Of the 37 CRVO eyes studied, 17 (45.9%) eyes had ischemic CRVO, 16 (43.2%) had non-ischemic CRVO and 4 (10.8%) eyes had conversion non-ischemic into ischemic CRVO during the follow-up period. Of the 81 BRVO eyes, 64(79.0%) had major, 17(21%) had peripheric BRVO. The classification regarding non-ischemic and ischemic CRVO and also major and peripheric BRVO was performed by using FA and ophthalmoscopy. The inner retinal ischemia signs of anterior and posterior neovascularization were such as; in 17 ischemic CRVO eyes 2(11.8%) had rubeosis iridis (RI), and 5 (29.4%) had NVG at the initial examination, 4(23.5%) had developed NVG within 7 months following the initial diagnosis. Posterior segment complications were such as optic nerve head (NVD) or retinal new vessels (NVE) were seen in 15(88.2%) of ischemic CRVO, and 4 (100%) of conversion

Non-ischemic into ischemic CRVO eyes within 7 months following the initial diagnosis. In major BRVO patients 5 (7.8%) had developed RI and 3(4.7%) had developed NVG within 7 months following the initial diagnosis. NVD/NVE were found in 15 (23.4%) of 64 major BRVO patients within 8 months following the initial diagnosis (Table 2).

Table 2: *Anterior and posterior segment complications in CRVO and BRVO eyes.*

	Ischemic CRVO		Nonischemic-Ischemic CRVO		Major BRVO	
	İnitial	Late	Initial	Late	Initial	Late
RI	2(11.8%)					5(7.8%)
NVG	5(29.4%)	4(23.6%)				3(4.7%)
NVD/ NVE		15(88.2%)		4(100%)		15(23.4%)

The time interval between onset of CRVO and examination may influence the VA considerably. 25 eyes with CRVO and 45 eyes with BRVO came to visit in the first 90 days of their complaints. Analysis of the data on VA in the three types of CRVO showed that initial VA 20/40 or better was found in 11.8% (2 eyes) of ischemic CRVO, 43.7% (5 eyes) of the non-ischemic CRVO group; between 20/50-20/100 was found in 11.8% (2 eyes) of ischemic CRVO and 31.3% (7 eyes) of the non-ischemic eyes and 100% (4 eyes) in conversion non-ischemic into ischemic CRVO eyes. The initial VA 20/200 or worse was found in 76.4% (13 eyes) of ischemic CRVO and 25% (4 eyes) of non-ischemic CRVO eyes. The final VA 20/200 or worse was found in 100% (17 eyes) ischemic CRVO, and 100% (4 eyes) in conversion non-ischemic into ischemic CRVO. Deterioration of VA in all three groups of CRVO eyes was due to CME and optic atrophy (Table 3).

In 64 major BRVO patients, eyes first seen ≤2 weeks after onset, with VA 20/40 and 20/50- 20/100 showed spontaneous VA improvement. In 11(17.2%) eyes the initial VA was 20/40, and in 25(39.0%) eyes the initial VA was 20/50-20/100. The initial VA was 20/200 or worse was found in 28(43.8%) eyes of major BRVO patients. The final VA 20/40 or better was found in 37.5% (24 eyes) of major BRVO, and; 20/50-20/100 was found in 21.9% (14 eyes). The final VA 20/200 or worse was found in 40.6% (26 eyes) of major BRVO patients due to the macular pigmentary

degeneration. The initial and final VA was 20/40 or better in all of the peripheric BRVO patients (Table 4).

Vision treated ocular complications in the early(within 90 days) and late(90-365 days) period were shown in (Table 5). The most prevalent ocular complication was ME in the early period, was seen in all types of CRVO and BRVO eyes (Table 5).

Table 3: *Visual acuity measurement in CRVO.*

	Ischemic CRVO		Non-ischemic CRVO		Non-ischemic CRVO –ischemic CRVO	
	İnitial	Late	Initial	Late	Initial	Late
20/40>	2(11.8%)	-	5(43.7%)	7(43.8%)	-	-
20/50-20/100	2(11.8%)	-	7(31.3%)	9(56.2%)	4(100%)	-
<20/200	13(76.4%)	17(100%)	4(25%)		-	4(100%)

Table 4: *Visual acuity measurements in BRVO.*

	Major BRVO (n= %)		Peripheric BRVO (n= %)	
	İnitial	**Final**	**Initial**	**Final**
20/40>	11(17.2%)	24 (37.5%)	17 (100%)	17 (100%)
20/50-20/100	25(39.0%)	14(21.9%)	-	-
<20/200	28(43.8%)	26(40.6%)	-	-

Table 5: *Vision treated ocular complications in CRVO and BRVO eyes.*

	ICRVO		Non-ischemic CRVO		Non-ischemic to ischemic CRVO		Major BRVO		Peripheric BRVO	
	Early	**Late**	**Early**	**Late**	**Early**	**Late**	**Early**	**Late**	**Early**	**Late**
ME	16 (94.1%)	8 (47.1%)	16 (100%)	3 (18.7%)	4 (100%)	3 (75%)	60 (98.7%)	26 (40.6%)	4 (23.50%)	-
PE degeneration								10 (15.6%)		
VH	1 (5.9%)	2 (11.8%)				1 (25%)	4 (6.3%)	9 (14.1%)		
Optic atrophy		7 (41.10%)		1 (6.30%)				1 (6.30%)		
No visual disturbance				12 (75%)				18 (28.1%)	13 (76.5%)	17 (100%)
Total	17 (100%)	17 (100%)	16 (100%)	16 (100%)	4 (100%)	4 (100%)	64 (100%)	64 (100%)	17 (100%)	17 (100%)

Discussion

RVO, is one of the most common cause of retinal vascular disease, and if left untreated, it can result in severe visual loss. It is more commonly seen in patients older than 65 years [8-10]. In the Blue Mountain Eye Study, the incidence was reported as 0.7% in patients younger than 60 years old, and 4.6% in patients older than 80 years old. Multiple studies have suggested that, when compared with women, men may be at increased risk of RVO. There are also inconsistent reports regarding differences in ethnic predisposition to CRVO, with finding of 58% increased risk in black patients. Detailed history taking, careful assessment for any complications and laboratory investigations of cardiovascular risk profiles, are important evaluations to be performed for RVO cases. Certain systemic diseases such as diabetes, hypertension and hyperlipidaemia are associated with increasing age. Studies have shown that advancing age, elevated blood pressure, hyperlipidaemia, elevated blood sugar and ocular perfusion were principal variables predicting incident RVO. Of these systemic risk factors, one meta-analysis found that 47.9% of RVO cases were attributed to hypertension, 20.1% to hyperlipidaemia, and 4.9% to diabetes mellitus. Systemic hypertension is a significant risk factor and accelerates arterial stiffness [11-13]. Some studies have found increased risk of cerebrovascular and cardiovascular disease in patients with RVO, including a greater risk of developing acute myocardial infarction after a diagnosis of RVO [14,15]. Other risk factors such as high body mass index, hypercoagulable state, and peripheral artery disease, different forms of vasculitis, neoplasm, oral contraceptives, stroke and smoking have also been reported. Some studies have revealed an association between RVO and hyper viscosity due to high haematocrit. Higher blood viscosity increases erythrocyte aggregation under conditions of low blood flow [16]. The association of RVO and smoking is explained by the inflammatory stimulus of smoking, although it's not certain. The

role of coagulation factors in the development of RVO remains unclear.

Although majority of cases have unilateral occlusion, 5-6% of BRVO and 10% of CRVO patients present with bilateral involvement [7-17]. In our study, CRVO and BRVO were seen more commonly in men older than 60 years. Systemic hypertension, hyperlipidaemia, and diabetes mellitus are significant risk factors. Smoking and alcohol consumption were found as 64.5% in men, 39.6% in women. Oral contraceptive was only found in 4.4% of patients. We think that it was associated with cultural reasons. All these results were harmonic with previous studies. Hyperopia and glaucoma have been reported as local ophthalmic risk factors [2]. There are differences in the role of each single risk factor in pathogenesis of CRVO and BRVO [18,19]. For example, hypermetropia, arteriosclerosis and high blood pressure are more common in BRVO, whereas raised intraocular pressure, leading to venous stasis in blood flow, is more common in CRVO. This demonstrates that CRVO and BRVO are different entities with different prognosis and management. It has been postulated that eyes with shorter axial length have smaller lamina cribrosa apertures and a narrower scleral canal through which the central retinal vein and artery passing, causing physical blockage in the vein which predisposes to thrombus formation [20,21]. Patients with CRVO were found to have more profound visual loss than patients with BRVO. This can be explained from the pattern of affectation of the retina by the vessel involved. Central retinal vein usually involves the four quadrants and inadvertently the posterior pole is affected, compared with when only a branch is involved which may likely spare the macular region. The visual prognosis for patients with ischemic and non-ischemic CRVO differs. The ischemic CRVO carries a poor prognosis as there is a high risk to develop macular edema, ischemic maculopathy, neovascularization and eventually rubeotic glaucoma. Reported that 51% of eyes with non-ischemic CRVO gained a VA 20/40 or better without any treatment [22,23].

BRVO has a good prognosis; 50–60% of eyes have been reported to have a final VA of 20/40 or better even without any treatment. A poor visual prognosis has been reported in patients with chronic macular edema or macular ischemia. Generally, it is difficult to determine visual prognosis for patients with BRVO in the acute phase of the disease. Patients with super temporal quadrant BRVO also experience greater degrees of VA loss relative to BRVO in other quadrants. The strongest predictors for NV of iris or angle were found to be visual acuity and extend of ischemic areas as seen on FA. 35% of ischemic eyes in the CVOS, developed NV of the iris or angle, compared to only 10% developing anterior chamber NV in non-ischemic eyes [5]. RI and NVG are severe complications of CRVO which occur in 12% to 30% of all cases. The stimulus for anterior segment neovascularisation is poorly understood but is thought to be related to severe retinal ischaemia. VA was a poor predictor of the development of RI, because in the early course of CRVO, as reported previous studies, vision was decreased owing to macular oedema as well as perifoveal capillary occlusion (ischaemia). RI developed in 12 of 57 patients (21%) with early CRVO within a period of 3 weeks to 7 months after the onset of symptoms, and in all these patients' angle neovascularisation and glaucoma ensued. RI correlated most directly with the extent of capillary no perfusion that was observed on the FA. RI was correlated also with the funduscopic observation of cotton-wool spots, but less well than with the extent of capillary occlusion. This was probably because cotton-wool spots became individually less distinguishable as they became more numerous and confluent. Rubeosis developed in 80-86% of eyes with an absent parafoveally net of 3 to 4 quadrants of posterior pole or peripheral capillary occlusion, but appeared in only 3-9% of those with an intact parafoveally net and less than 1 quadrant of capillary occlusion. NV of retina or disc secondary to an initially non-ischemic CRVO was found in up to 33% over a period of up to 15 month. As for ischemic CRVO, the incidence of NV was up to 20% over a period of 9 month. In some studies with no sub-division, NV was seen in up to 50% of patients after a 6 month period. Ischemic CRVO is associated with NVG in 23%-60% of cases, and is first detected by gonioscopy [24].

Macular edema (ME) is the main complication in RVO patients and is closely associated with retinal hypoxia, and the degree of hypoxia in the centre of the macula corresponds to the decrease in VA [5]. ME is a major complication of CRVO and associated with poor visual prognosis without treatment. Early treatment is essential since the longer the edema exists, the worse is the structural damage to the fovea, but even late treatment could improve VA. In cases of ischemic CRVO resolution of ME ranged up to 73% in up to 15 month, compared to the non-ischemic type where the corresponding proportion was about 30% by 15 month. The incidence of vitreous haemorrhage (VH) in CRVO patients was described in one study and was 10% in a 9 month follow-up. The incidence of NV is believed to be relatively low but there is no meaningful data on BRVO in relation to NV and endovascular glaucoma (NVG) [5]. ME in BRVO patients develops in 5%-15% of eyes in 12 mo [5]. The extent of macular or foveal involvement in acute BRVO is an important factor in determining the prognosis [24]. Patients with retinal ischemia of at least 5-disc diameters in size have 36% chances of developing VH if laser photocoagulation is not perforemed. It is important to note that the Branch Vein Occlusion Study was conducted in the mid-1980s, and patients waited 3 months to allow macular edema to resolve before laser treatments; whereas today pharmacological treatments may commence immediately to bridge that gap [24].

In our study, FA provided information on retinal capillary non perfusion. Ischemic CRVO can be primary or progression of non-ischemic CRVO, although progression is not common. Ischemic CRVO has a much lower visual prognosis and accounts for about 46% of cases. Around 94% of patients with visual acuities worse than 20/200 have ischemic CRVO. Ischemic CRVO carries poorer prognosis and is defined as having at least 10 disc areas of retinal capillary no perfusion. If the CRVO does not become ischemic, return to baseline or near baseline vision occurs in about 43.7% of patients. Chronic ME is the main cause of poor vision. In most case the prognosis correlates with initial VA. If VA is 20/40 or better, the VA is likely to remain the same. If the patient has 20/50-20/200 vision, the clinical course varies. VA may improve, stay the same, or worsen. In VA worse than 20/200, improvement is unlikely. Ischemic CRVO has a more variable prognosis due to macular ischemia. Patients have a high risk of NVG due to the development of RI in 52.9% of eyes, usually between 2 to 6 months. Retinal neovascularization occurs in 60% of eyes. In BRVO patients, 40.6% of eyes have VA worse than 20/200 due to macular edema. Retinal neovascularisation (NVD/NVE) occurs in 28.1% of BRVO eyes. Anterior segment complications occurs in short time when compared to posterior segment complications.

Conclusion

RVO is a common cause of retinal vascular disease; and if left untreated, it can result in severe visual loss. Recognizing the clinical features of RVO and promptly diagnosing treatable causes of visual morbidity, including macular edema and neovascularization, can result in improved clinical outcomes and often restoration of visual acuity. In non-ischemic CRVO, initial follow-up should at 3 months, although the patient should return sooner if the vision deteriorates. Patients with ischemic CRVO should be monitored on a monthly basis for 6 months for the development of anterior segment neovascularization or neovascular glaucoma, with gonioscopy performed at each visit prior to dilation. Patients should be monitored for up to 1 year to assess for significant ischemia and macular edema.

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Declaration of Interest

The author has no relevant affiliations or financial involvement with a financial interest in or financial with the subject matter or materials discussed in the manuscript.

Statement of Ethics

The study prothocol was approved by the Ethics committee of Kartal, Dr. Lutfi Kirdar Education and Training Hospital, Istanbul, Turkey (decision number: 2020/514/173/2).

Conflicts of Interest

There is no conflict of interest.

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