Current Imaging and Treatment Approach in Retinal Arterial Macroaneurysm Series: A Single-Center Retrospective Study

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ABSTRACT

Aim: We aimed to evaluate the current imaging findings and treatment approaches for retinal artery macroaneurysm (RAM).

Methods: Our study was a retrospective single-center observational study of 26 RAM cases between 2018 and 2023. All patients underwent a full ophthalmoscopic examination. RAMs have been classified as hemorrhagic, exudative, or inactive (quiescent) based on foveal involvement. Treatment methods, complications, and final best-corrected visual acuity (BCVA) were evaluated.

Results: Among the cases, 57.7% were women, and the average age of all patients was 67.9 ± 14.6. The follow-up period was 33.5 ± 15.4 months. When evaluated in terms of the classification of RAMs, 12 cases (52.1%) were inactive RAM, 10 cases were exudative RAM (38.5%), and 4 cases (15.4 %) were hemorrhagic. Of the 12 inactive RAM cases, while 9 cases were followed, laser treatment was applied in 3. At least three doses of intravitreal anti-vascular endothelial growth factor (VEGF) were administered to 8 of the 10 exudative RAM cases. Two of the four hemorrhagic RAM cases were treated with pars plana vitrectomy (PPV), one was treated with intravitreal anti-VEGF, and only one was followed up. A significant increase in the final visual acuity was found in all patients, regardless of treatment.

Conclusion: Although observation was a good choice in quiescent RAM cases without foveal involvement, anti-VEGF treatment, PPV, and laser photocoagulation were effective treatment methods in RAMs with foveal involvement.

Key Words: Imaging, retinal arterial macroaneurysm, treatment.

INTRODUCTION

Retinal arteriolar macroaneurysm (RAM) refers to the acquired dilatation of the branches of the central retinal artery. Frequently, it develops at the second or third bifurcation of the temporal retinal artery [1,2] Though RAM cases may be asymptomatic, they might cause a decrease in visual acuity, resulting in macular edema, serous detachment, hard exudate accumulation in the macula, retinal vein branch occlusion, and pre-macular or vitreous hemorrhage. [3] RAMs have been classified as inactive (quiescent), hemorrhagic, or exudative. [4] Inactive RAMs are asymptomatic and may be complicated by hemorrhage or exudate. Exudative RAMs cause edema or exudate in the macula, and hemorrhagic RAMs may cause a decrease in visual acuity with hemorrhage in the premacular or vitreous. [5]

Thickening on the vessel wall, fibrin, and macrophage has been established histologically in the RAM area. It is thought that RAMs develop with aging and arteriosclerotic processes, in addition to high blood pressure. [6] It has been reported that hemorrhagic RAMs are more frequently associated with hypertension than exudative RAMs, and hypertension causes rigidity and dilatation of the vessel wall, and it is asserted that hemorrhage develops with the

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impact of increased pressure and pulsation. [4,6] Some studies have reported spontaneous resolution of RAMs [1,5,7] and observation in cases with cystoid macular edema and hard exudate in the macula may result in permanent damage. [7] Argon laser photocoagulation for the treatment of RAM was first proposed by Gass. [8] It has been reported that direct laser treatment of RAM shortened the resolution period of the lesion, but treatment applied to the area surrounding RAM reduced the risk of exudative complications, reducing the oscillation of angiogenic factors and inflammatory cytokines. [3] But it is announced that laser treatment is associated with some complications, such as occlusion of the retinal artery and RAM rupture. [2] Anti-VEGF agents have been effective in inhibiting the formation of abnormal vessels and VEGF mediative vessel permeability. [8] Anti-VEGF treatment is an alternative treatment choice for exudative RAMs. [2,8] RAMs causing pre-macular or vitreous hemorrhage have been treated with pars plana vitrectomy and ILM peeling, pneumatic translocation, sub-retinal blood drainage, or injection of recombined plasminogen activator or a combination of all.[1]

This study aimed to retrospectively analyze the characteristics of the clinical features and applied treatment methods of 26 patients with RAM in our ophthalmology department.

MATERIAL AND METHOD

This was a retrospective observational study conducted according to the principles of the Declaration of Helsinki. The study protocol was approved by the Ethical Committee of Clinical Research of Ankara Etlik City Hospital. Each patient signed an acknowledged consent form between 2018 –2023. The demographic data included age, sex, and race. Exclusion criteria were previous ocular diseases, history of vitreoretinal surgery, Leber miliary aneurysm, idiopathic retinal vasculitis, aneurysm of neuroretinitis, polypoid choroidal vasculopathy, and branch retinal vein occlusion. We considered whether the patients had systemic diseases, especially essential hypertension or not was pondered.

The patients underwent a full ophthalmologic examination, which included best-corrected visual acuity, slit-lamp examination, and dilated fundus examination. A color fundus photography, fluorescein angiography ((TRC 50DX IA, Topcon, Tokyo, Japan), Optical Coherence Tomography (OCT) Spectrails HRA + OCT; Heidelberg Engineering, Heidelberg, Germany), and Optical Coherence Tomography Angiography (OCTA) AngioVue (Optovue Inc, Fremont, CA) have been performed and analyzed. RAMs were classified as hemorrhagic, exudative, or inactive based on clinical imaging results. Argon lasers have been applied to cases with leaks around the RAM. Intravitreal anti-VEGF was applied under sterile conditions in patients with foveal intra/subretinal fluid and hard exudate. Pars plana vitrectomy was performed in cases with intense vitreous and premacular hemorrhage that was not reabsorbed. The absence of sub-retinal fluid or hemorrhage in OCT and FFA or regression of RAM in fundus examination was considered functionally inactive RAM. At the same time, the absence of flow in the macroaneurysm area on OCTA is considered a sign of closure.

Visual acuity data were transformed into logarithm of the minimum angle of resolution (logMAR) for statistical analysis. Statistical analyses were performed using SPSS version 21 (IBM, Chicago, IL, USA) and nonparametric paired sample statistics with Pearson correlations and significance set at a P value of 0.05.

RESULTS

The mean age of the patients was 67. 9± 14.6 (range, 28–95) years; 15 (57.7%) were female and 11 (42.3%) were male, and all patients were Caucasian Turks. The average follow-up period was 33.5± 15.4 months (12 – 60 months).

The average visual acuity of the patients at the first examination was logMAR 0,70±0.8 (0,0 – 3,1). In 12 cases, the visual acuity was 20/20 at 20/50, in three patients by 20/60 at 20/100, in eleven cases at 20/100, and worse. The average visual acuity of the cases at the last examination was LogMAR 0,36±0,4 (0,0 – 0,5). While visual acuity in 15 of the cases significantly increased ($p \leq 0,05$), there was no change in visual acuity in 11 cases. In 17 (65.4 %) cases, RAMs were observed in the left eye (n = 17). Four patients had hemorrhagic RAM, 10 had exudative RAM and 12 had inactive RAM. Fourteen patients (53.8%) had history of essential hypertension.

Macular hemorrhage was established in four patients. (Table 1) Visual acuity was 20/2500, 20/400, 20/2500, and 20/250, respectively. In the first three cases, a sub-ILM hemorrhage on the OCT section was established. Pars pla-

Table 1: *Clinical data of the patients with hemorrhagic RAMs*

No	Age (years)	Sex (F/M)	Side (R/L)	VA_{init}	VA_{end}	Follow-up (months)	Treatment	Type (E/H/Q)	Location
H1	78	M	L	20/2500	20/40	17	VRC	H	Superotemporal
H2	59	F	L	20/400	20/50	44	VRC	H	Superotemporal
H3	78	F	R	20/2500	20/640	42	Observation	H	Superotemporal
H4	63	M	R	20/250	20/60	36	IVI	H	Inferotemporal

E, presence of exudation at the macula; H, presence of hemorrhage at the macula; Q, quiescent which indicates no exudation or hemorrhage at the macula; R, right eye; L, left eye; F, female; M, male; IVI, intravitreal injection.

na vitrectomy, posterior hyaloid removal, and ILM peeling were performed in cases H1 and H2. (Figure 1) The final visual acuity of the patients who underwent vitrectomy was 20/40 and 20/50. No postoperative complications developed after the vitrectomy. Subretinal hemorrhage was observed in the macula and around the RAM at the first examination in case H3. After 42 months treatment-free observation, the final visual acuity was 20/640. Visual acuity of a patient (case H4) who received 3 doses of bevacizumab improved to 20/60. The first BCVA of hemorrhagic RAM cases was established as 1,87±1,5 (logMAR, final BCVA was established as 0,72±0,1 (logMAR) ($p>0,05$). (Table 1)

A yellow exudative ring was observed around the aneurysmal dilatation fundoscopically in seven of the exudative cases. The serous detachment was accompanied by cystoid edema on OCT in three exudative cases (cases E2, E8, and E10). The laser was applied around and on the aneurysm in three exudative cases. One case (case E1) had intense exudate in the macula, and a laser was applied onto the leaking aneurysm. At the end of 5 years of follow-up, the patient's visual acuity increased from 20/640 to 20/250. Significant photoreceptor loss was observed in the outer retinal layers on the final OCT image of the patient. (Figure 2) (Table 2)

Intravitreal anti-VEGF was administered in 8 exudative cases. In one of the cases (case E3), laser photocoagulation was applied together with anti-VEGF, and the final visual acuity was measured as 20/33. For all cases in which anti-VEGF was applied, three doses of bevacizumab were administered. After three doses of intravitreal bevacizumab, two doses of intravitreal dexamethasone implant in 1 case, two doses of intravitreal ranibizumab in 1 case, and three doses of intravitreal aflibercept in 3 cases were administered. The average baseline BCVA of exudative RAM cases was 0,9±0,3 (logMAR). Following treatment, the final average BCVA was measured as 0,51± (logMAR) ($p<0,05$). (Table 2)

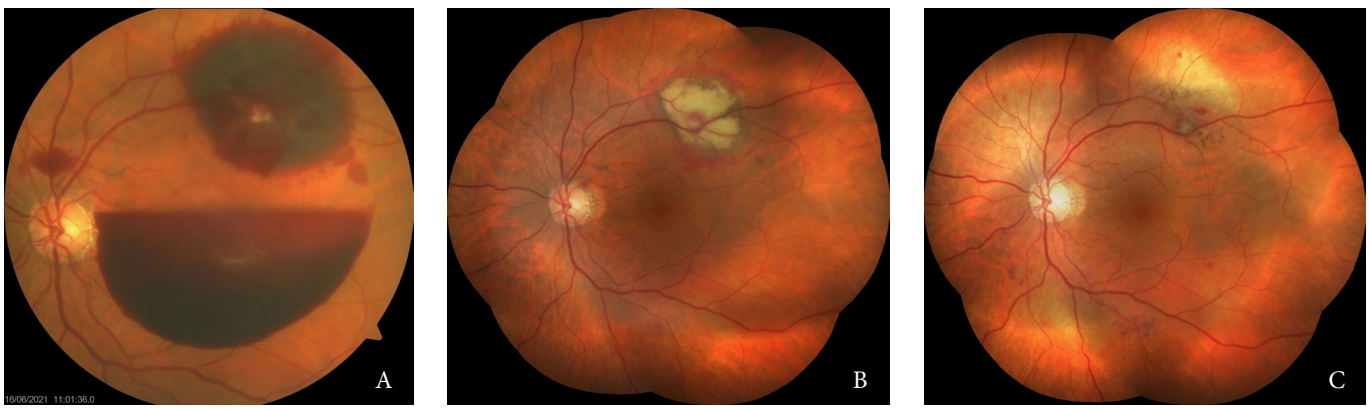

Figure 1: *Color fundus photography (A) of left eye at baseline reveals superotemporal RAM with subhyaloidal hemorrhage at the macula; (B) dehemoblobinized hemorrhage around the RAM and normal macula at postoperative 1st week; (C) laser scars around the RAM at sixth month. (Case H1)*

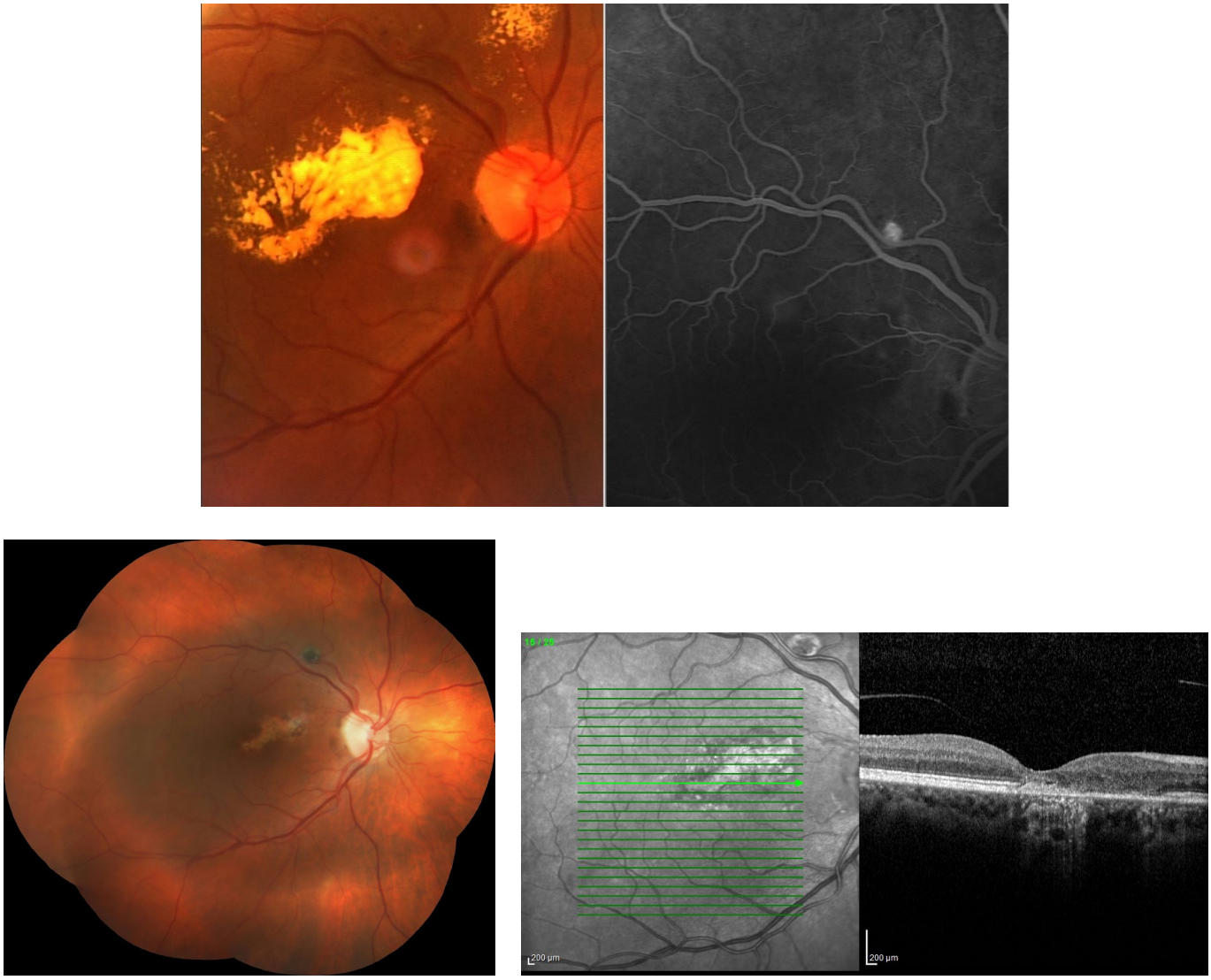

Figure 2: *Color fundus photography (A) of the right eye at baseline shows extensive exudate at macula and fluorescein angiography indicates hyperfluorescence on the RAM. At final visit (B) color fundus photography reveals macular scar and closed RAM. OCT image shows (C) loss of outer retinal layers at last visit. (Case E1)*

Twelve patients had inactive RAMs. While 9 of the inactive RAMs were observed, laser photocoagulation was applied to three of them. Although asymptomatic, the reason why the laser was applied in three cases was the observation of leakage around the RAM. This was performed to prevent leakage into the macula. (Table 3) While the first average BCVA of inactive RAM was 0,15±0,2 (logMAR), the average of the final BCVA was determined as 0,12±0,1 (logMAR) (p>0,05).

Direct and indirect laser photocoagulation were performed for all cases in which laser photocoagulation was applied. The first and final best-corrected visual acuity (BCVA) in cases with photocoagulation was consecutively 0,47±0,64 and 0,37±0,44. No complications were observed in the patients who underwent photocoagulation. In 16 cases (61.5%), RAM was superotemporal, 8 (30.8%) were inferotemporal, and 2 (7.7%) were superonasal. On fluorescein angiography, RAM was observed as nonhomogenous hyperfluorescence on the arterial trace in the earlier phase. Hyperfluorescence increased at the latest phase, and leakage was observed in the macula in patients with macular edema. Hypofluorescence was observed due to window defects depending on hemorrhage in hemorrhagic RAMs. (Figure 3) Optic coherence tomography angiography images showed flow in non-thrombosed RAMs, while no flow was observed in thrombosed RAMs. (Figure 4)

Table 2: *Clinical data of the patients with exudative RAMs*

No	Age (years)	Sex (F/M)	Side (R/L)	VA_{init}	VA_{end}	Follow-up (months)	Treatment	Type (E/H/Q)	Location
E1	72	F	R	20/640	20/250	60	Photocoagulation	E	Superotemporal
E2	82	F	L	20/100	20/100	33	Photocoagulation	E	Inferotemporal
E3	81	F	L	20/60	20/33	13	Photocoagulation+IVI	E	Superotemporal
E4	84	M	R	20/66	20/20	39	IVI	E	Superotemporal
E5	69	F	R	20/160	20/160	17	IVI	E	Inferotemporal
E6	74	F	R	20/250	20/160	21	IVI	E	Superotemporal
E7	62	F	R	20/250	20/160	57	IVI	E	Superotemporal
E8	37	M	L	20/250	20/40	57	IVI	E	Inferotemporal
E9	72	M	L	20/400	20/60	49	IVI	E	Inferotemporal
E10	68	M	L	20/40	20/40	21	IVI	E	Superotemporal

E, presence of exudation at the macula; H, presence of hemorrhage at the macula; Q, quiescent which indicates no exudation or hemorrhage at the macula; R, right eye; L, left eye; F, female; M, male; IVI, intravitreal injection.

Table 3: *Clinical data of the patients with quiescent RAMs*

No	Age (years)	Sex (F/M)	Side (R/L)	VA_{init}	VA_{end}	Follow-up (months)	Treatment	Type (E/H/Q)	Location
Q1	69	F	L	20/20	20/20	38	Photocoagulation	Q	Superotemporal
Q2	53	M	L	20/25	20/22	25	Photocoagulation	Q	Inferotemporal
Q3	68	M	L	20/22	20/22	15	Photocoagulation	Q	Inferotemporal
Q4	83	F	L	20/60	20/40	12	Observation	Q	Superotemporal
Q5	72	F	R	20/22	20/22	25	Observation	Q	Superotemporal
Q6	78	F	R	20/28	20/25	49	Observation	Q	Inferotemporal
Q7	95	F	L	20/40	20/40	56	Observation	Q	Superotemporal
Q8	28	M	L	20/20	20/20	27	Observation	Q	Superotemporal
Q9	58	M	L	20/20	20/20	18	Observation	Q	Superotemporal
Q10	70	F	L	20/22	20/22	24	Observation	Q	Superonasal
Q11	48	F	L	20/20	20/20	50	Observation	Q	Superotemporal
Q12	65	M	L	20/20	20/20	27	Observation	Q	Superonasal

E, presence of exudation at the macula; H, presence of hemorrhage at the macula; Q, quiescent which indicates no exudation or hemorrhage at the macula; R, right eye; L, left eye; F, female; M, male; IVI, intravitreal injection.

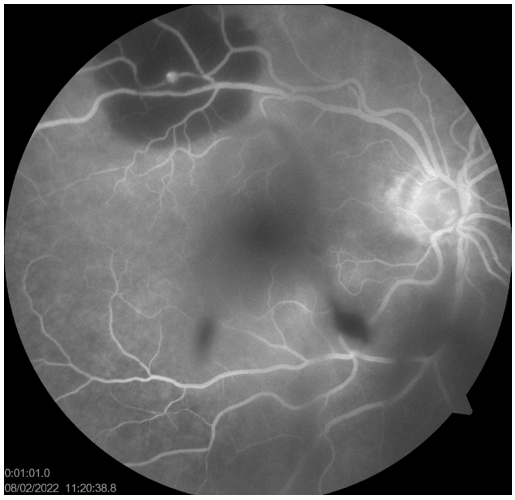

Figure 3: *Fluorescein angiography shows RAM as a localized arterial dilatation and hypofluorescence due to window defect depending on hemorrhage around the RAM. (Case E7)*

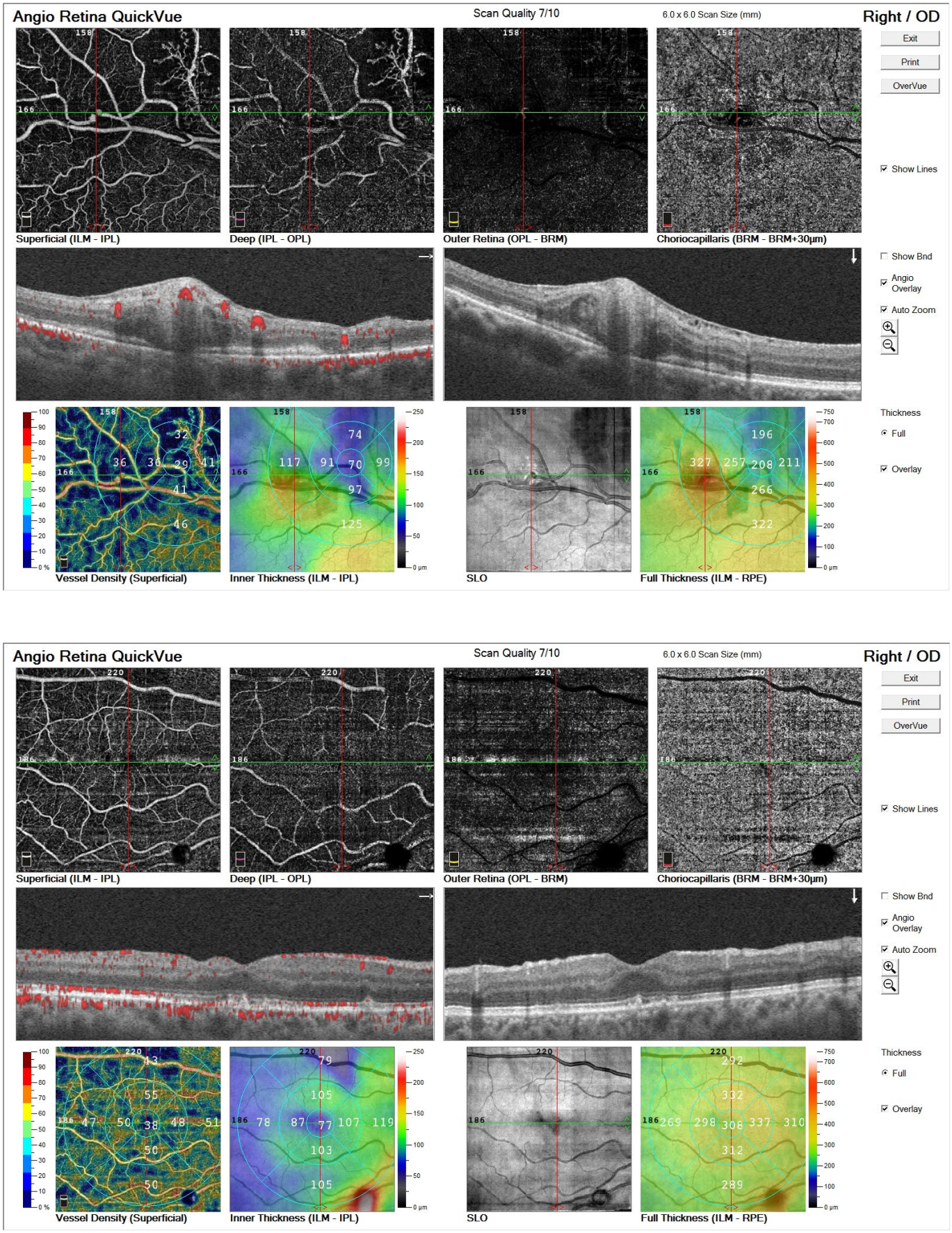

Figure 4: *(A) OCT angiography deep and superficial capillary plexus segmentation (case E7) reveals flow within the RAM and (B) OCT angiography of case Q6 shows no obvious flow within the RAM.*

DISCUSSION

This case series included 26 patients with RAM. The patients were followed for approximately 33 months. Vitrectomy was performed in two cases, laser photocoagulation in 6, and intravitreal anti-VEGF injection in 8 cases. Ten cases were only observed. Visual acuity increased in 15 patients, but there was no change in 11 patients.

Retinal artery macroaneurysms are acquired vessel dilatations that frequently form in the second and third branching areas of the retinal artery. Known risk factors for RAM include female sex, hypertension, atherosclerosis, hyperlipidemia, older age, polycythemia, and cerebrovascular disease. [9] In our retrospective study, in most cases, female sexuality, a history of hypertension, and older age (> 60 years) (57.7%, 53.8%, and 76.7%, respectively) were consistent with the literature.

Although there is no consensus on RAM treatment, the vast majority of RAMs resolve spontaneously. Nonetheless, in symptomatic cases with significant exudation and hemorrhage, observation may result in irreversible damage to photoreceptors. Laser photocoagulation has been used for RAM treatment for many years. [10] In a study by Brown et al., the laser and observation results of RAM cases were compared. At the end of the study, the laser that was directly applied to the macroaneurysm did not have a meaningful positive impact on the demonstrative results compared to the observation group. [10] In a series of twenty-seven cases, fourteen cases were followed up without treatment, and laser treatment was applied in 13 of them. It was emphasized that laser treatment was effective in RAMs with hemorrhagic components. [3] In patients with intensive hyaloidal or retinal hemorrhage, vitrectomy should be performed urgently, and ILM peeling, anti-VEGF and/or tissue plasminogen activator, and intravitreal gas injection should be applied during vitrectomy according to the condition of the patient. [1]

It was also reported that direct application of laser onto macroaneurysms shortened the closure of the lesion, and application to the surrounding area would reduce possible exudation, decreasing angiogenic factors and inflammatory cytokines. [3,4] A longer period (200-500 milliseconds), larger laser spot size (200 – 500 µm), and minimal power (100 – 300 mW) were recommended to obtain a slight laser burn. However, attention should be paid to retinal artery occlusion when applying laser directly to retinal artery. In the indirect laser, shorter periods (100-200 milliseconds), mean weight (400 mW), and mean spot dimension (200µm) were applied around the aneurysm. [11.12] Laser photocoagulation may cause sight-threatening complications including arteriolar occlusion, retinal tear, shunt from artery to a vein, sudden bleeding, loss of capillary perfusion, formation of choroid neovascularization, elongation of exudation, traction in the retina, scarring in the sub-retinal area and symptomatic scotoma.[10] In our case series, laser photocoagulation was used in 6 RAM cases. While half of the cases in which photocoagulation was applied were asymptomatic, half of the cases were exudative. In two cases who were exudative and for whom only laser was applied, the final visual acuity was under 20/100.

Although the mechanism of the effectiveness of anti-VEGFs in the treatment of RAMs-related macular edema is not clearly known, there are some hypotheses. Localized ischemia and VEGF up-regulation in the aneurysmal area cause VEGF associated vascular permeability and dilatation, which result in macular edema and hard exudate.[5] It has been put forward that there were two possible mechanisms to explain how anti-VEGF agents accelerated the resolution of exudative and hemorrhagic RAMs. Intravitreal anti-VEGF agents eliminate the angiogenic and vascular permeability effects of VEGF. VEGF increases nitric oxide levels, which has a vasodilatory effect. Anti-VEGF agents exert a vasoconstrictive effect by decreasing nitric levels. Vasoconstriction has been reported to decrease macular edema, regardless of vasopermeability. In the second mechanism, VEGF activates the coagulation cascade. Anti-VEGF agents may be effective in breaking down the equilibrium between the coagulation and fibrinolysis processes. Thus, these agents have an accelerating effect in clearing off retinal and vitreous hemorrhage. [13]

In their series composed of 37 cases, Pinchi et al. indicated that edema in the 6 mm dimensional para-foveal area disappeared 4 weeks following 3rd injection, and exudations and edema in the para-macular area also disappeared gradually. [14] In a retrospective study of 49 hemorrhagic RAM cases, intravitreal bevacizumab was administered in 33 cases, and 16 cases were followed up. Nineteen cases of hemorrhage in the fovea were evaluated in the intraretinal hemorrhage (IRH) group. Thirteen patients with intraretinal

hemorrhage were treated with intravitreal bevacizumab, and 6 of them were followed up. No significant difference was found in the final BCVA between treated and untreated cases. [15] In our case series, anti-VEGF was administered in only one hemorrhagic case. The hemorrhage resolved following three doses of bevacizumab, and the final visual acuity increased to 20/60. In a study in which one group was treated with bevacizumab and the other was not been treated, recovery was faster in visual acuity in the bevacizumab group. There was not a meaningful difference in vision and thickness of macula between two groups. [13] In a study in which ten cases with symptomatic RAM were treated with three doses of ranibizumab, it was reported that there was a significant exudate regression in eight cases. [16]

In the literature, there is a case report with familial retinal arterial macroaneurysm who was applied dexamethasone implant for macular edema. It has been reported that intravitreal dexamethasone implant had marked visual and structural outcomes for persistent macular edema poorly responsive to intravitreal injections.[17] In our case series, there is a case with macular edema who was applied dexamethasone implant after 3 doses of bevacizumab injection. We also obtained good visual improvement after steroid injection in our RAM case.

Corticosteroids contribute to reduce macular edema by inhibiting macrophages that release angiogenic factors and reducing major histocompatibility complexes. Additionaly, they alter the composition of endothelial basal membrane by suppressing basement membrane dissolution and strengthening zonula ocludens to decerase permeability and leakage. [18,19]

In our observation group, twelve of our patients had inactive RAM, and their first visual acuity was 20/60 to 20/20. One patient had hemorrhagic RAM and left to spontaneously close because of bleeding around the RAM; the first visual acuity was 20/2500, and the final visual acuity remained 20/640 at the end of the follow-up. In the case of hemorrhagic RAM who had followed, we found that elongated macular hemorrhage ended with photoreceptor damage.

In most of our cases in the observation group, visual acuity was good, RAM was in a stable condition, and good results were obtained at the end of the follow-up. There was no significant increase in final visual acuity in the observation group because the fovea was not affected and their first visual acuity had already been good. It should be kept in mind that stable RAM cases would rupture spontaneously during the follow-up period, causing hemorrhagic and exudative complications, and should be closely followed up. [20]

The flow in the RAM can be imagined using OCTA. By determining and monitoring the flow in RAM in en-face OCTA, it is possible to measure the dimensions of RAM and make a diagnosis and follow-up. RAMs that are thrombosed are understood to have no flow on OCTA. [21] OCTA is superior in that it indicates 3-dimensional localization and flow in various retinal layers. Leakage around the RAM cannot be observed in OCTA. It does not provide information about the constitutional and morphological characteristics of RAMs. This condition should be considered for semi-thrombosed RAMs. [22] Though these limitations, there are case series with OCTA that are useful for diagnosis and follow-up. [21-23]

Our study had some limitations. The first limitation is that it has a retrospective and small sample size and does not have a control group. However, reaching a larger sampling number is difficult because of the rare nature of RAM. The second limitation is that we could not statistically compare the functional results of the treatment methods because the applied treatment methods are numerically inequivalent. Our study is in quality that it demonstrates the long-term results of RAM cases that were followed up and treated in certain years.

As a result, in our study, it was established that there was a statistically significant improvement in visual acuity at the end of the 33-week follow-up of our cases, independent of the treatment choice. In the three groups for whom laser photocoagulation, observation, and intravitreal anti-VEGF were used, we established that there was a significant increase in the first and final visual acuity in the anti-VEGF group. In the treated group, the first visual acuity was worse than in the observation group. It should be kept in mind that in cases of macular hemorrhage and macular edema, permanent photoreceptor damage may result if treatment for macular pathology is not applied.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent was obtained from all individual participants included in the study

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